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Use of Para-Aminosalicylic Acid in Chronic Pulmonary Tuberculosis*

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Introduction

In 1940, Bernheim[1] made the interesting observation that the oxygen consumption of tubercle bacilli was increased by salicylic and benzoic acid. Lehmann[2,3] studied various derivatives of benzoic acid with the purpose of discovering a substance possessing bacterio-static properties against the tubercle bacillus and found that para-aminosalicylic acid (PAS) was the most effective. Demonstration that the drug produced an inhibitory effect in vitro was confirmed by Sievers[4] and by Youmans,[5] and a retarding effect on the development of experimental tuberculosis was shown for mice by Youmans[5] and for guinea pigs by Feldman, Karlson and Hinshaw.[6] Trial of the drug by Lehmann and his collaborators[2,3,7,8] in chronic pulmonary tuberculosis indicated that a favorable clinical effect was produced on the symptoms of the disease, especially cough, expectoration and fever. No serious toxic effects of administration of para-aminosalicylic acid were reported by the Swedish investigators who have used the drug for three years. The side effects consisted of nausea, vomiting and transient albuminuria.

The effectiveness of PAS was studied in vitro and animal experiments, as well as its clinical use in a small series of cases of chronic pulmonary tuberculosis.

The purpose of Part I of this paper is to describe the inhibitory

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action of serum drawn from normal individuals and patients with pulmonary tuberculosis, following the ingestion of para-aminosalicylic acid on cultures of the tubercle bacillus. The clinical results of PAS are presented in Part II.

Part I — Laboratory Data

Method: The organism used in this study was H37Rv although similar results were obtained with other organisms isolated from patients with pulmonary tuberculosis.

Blood was drawn from patients as well as the normal subjects before, and from 1 to 4 hours after, taking the PAS by mouth. The serum was removed and diluted in Dubos media in various concentrations, with a final volume of culture medium of 5 cc. The concentration of serum in the Dubos media ranged from 1:2 to 1:100, the dilution most commonly used being from 1:10 to 1:50. A 7 to 9 day culture of H37Rv in Dubos media was employed, with an inoculum of 0.1 cc., equal to a dry weight of bacteria of 0.01 to 0.02 mg.

Results: In the accompanying table (Table I) the inhibitory effects of human serum drawn after ingestion of PAS is demonstrated. It will be seen that control serum had no effect on the growth of the tubercle bacillus, whereas 2 gms. of PAS exerted a definite bacteriostatic effect which was more marked with the smaller inoculum, 0.01 mg. This effect was observed in dilutions of 1:10, 1:15, 1:20, 1:30, 1:40 and 1:50. A more striking inhibition, lasting for the full 4 weeks' culture period, is demonstrated as the result of the larger doses, namely 3 to 4 g. of PAS.

Transplants made from the serum treated culture tubes showed an inhibition of growth in many instances in fresh Dubos media *without* additional serum. Considerable variation in these results took place, some tubes finally manifesting a heavy growth at the end of 4 weeks whereas others showed very slight growth.

Experiments were also conducted in which cultures were made with PAS in atmospheres containing 5 to 10 per cent oxygen. Although the growth of the tubercle bacillus in Dubos media was definitely inhibited when placed in atmospheres containing 5 and 10 per cent oxygen, as has been reported with other media previously,[9-11] no additive effect of the combination of anoxia and PAS was observed. No specific effect of increased oxygen concentrations between 40 and 60 per cent on cultures of the tubercle bacillus was noted except that the presence of 100 per cent oxygen exercised a retarding effect on growth for the first week. In earlier studies it has been reported that residence in 50 per cent oxygen atmospheres resulted in prolongation of life of infected rabbits without reduction in the extent of the pulmonary lesions,[12]

whereas exposure to 10 per cent oxygen exerted a retarding effect on the development of tuberculosis in guinea pigs.[13]

Lehman has stated that the bacilli in patients who have been treated with PAS do not develop resistance to this drug. In our in vitro studies of para-aminosalicylic acid, a concentration of 0.1 mg. per cent in 5 cc. of Dubos' media was adequate to inhibit a relatively large inoculum such as 0.02 mg. dry weight of the bacteria, when strain H37Rv was employed. A slight growth was manifested in cultures of this organism up to a concentration of 0.078 mg. per cent of para-aminosalicylic acid, with complete inhibition at higher concentrations of the drug. A similar response to PAS was observed when other strains of tubercle bacilli were used, isolated from three patients who had not been treated with the drug. In one patient, however, who had received a total dosage of 210 gms. of PAS, the organisms tested after treatment were resistant to 2.5 mg. per cent, and in two patients, who had received 630 gms. and 1000 gms. of PAS, the organisms were found to be resistant to 0.63 mg. per cent. Although Lehmann[3] stated that patients treated with PAS do not develop resistance to the drug, it would appear that strains either way vary considerably in their sensitivity to PAS or that some degree of resistance did develop in three of our patients. To decide this question, the sensitivity of the organism to PAS in vitro should be determined prior to and after each period of therapy.

Although the in vitro studies suggest that this drug is not as powerful a bacterio-static agent as streptomycin, our experiments in animals, including guinea pigs and rats, have also confirmed the reports of others that this drug has a definite retarding effect on the development of experimental tuberculosis in these animals.

Part II — Clinical Data

Methods: The dosage of the drug was similar to that employed by Lehmann and his collaborators.[2-4] Ten to eleven gms. were administered in three to five doses daily for three consecutive weeks, with a free interval of one week. One or more courses were given depending upon the tolerance of the patient to the drug. The urine was examined and the serum, non-protein-nitrogen (NPN), was determined before, during and after the completion of one or more courses of PAS. Other routine laboratory procedures, such as sedimentation rate, blood count and chest x-rays, were carried out before and at various periods following administration of the drug.

Results: Twelve patients with chronic pulmonary tuberculosis were treated. In five cases the paucity of symptoms of activity rendered estimation of the clinical value of the drug difficult.

TABLE I

INHIBITION OF TUBERCLE BACILLUS CULTURES DUE TO ADDITION OF HUMAN SERUM DRAWN AFTER INGESTION OF PAS*

Case No.		Inoculum H37Rv mg.	Dosage PAS gm.	Growth with Serum Before PAS	Growth with Serum After PAS 1 hr.	2 hrs.	3 hrs.	4 hrs.	REMARKS
1.	Normal	.01	3	++++	0	0	0		No growth after 4 wks. incubation.
2.	Normal	.01	3	++++	0	0	0		No growth after 4 wks. incubation.
3.	Normal	.01	3	No test	0	0	0		No growth after 4 wks. incubation.
4.	Patient	.01	3	No test	0	0	0		No growth after 4 wks. incubation.
5.	Normal	.02	2	++++		++		++++	Growth + after 1 wk., ++ after 3 wks., ++++ after 4 wks. on 2 hr. blood.
6.	Normal	.02	2	++++		++		++++	Growth + after 1 wk., ++ after 3 wks., ++++ after 4 wks. on 2 hr. blood.
7.	Normal	.02	2	++++		++		++++	Growth + after 1 wk., ++ after 3 wks., ++++ after 4 wks. on 2 hr. blood.
8.	Normal	.02	3	++++	+	+			Growth + after 4 wks. incubation.
9.	Normal	.02	4	++++	+	+			Growth + after 4 wks. incubation.
10.	Patient	.02	4	++++	+	+			Growth + after 4 wks. incubation.

*Cases 1 through 7 were tested with serum dilutions 1:10 to 1:50; Cases 8, 9 and 10 with serum dilutions 1:5 to 1:15, in Dubos media. One plus, +, indicates slight growth, ++ moderate growth, ++++ heavy growth.

These patients are included largely as a demonstration of the lack of toxic effects of PAS, but it was noted that decrease in expectoration occurred in four cases. No serious toxic effects of the drug were noted in any of this group who were in general good health, despite the presence of cavitary tuberculosis.

The remaining seven patients suffered from advanced pulmonary tuberculosis, in an active stage of the disease, with conspicuous symptoms. Streptomycin had been previously employed in four, with a temporary decrease in cough and expectoration, and improvement in general well being. In all of these seven seriously ill patients, decrease in cough and expectoration took place during the first week of treatment. At the end of the first course of three weeks' treatment, cough and expectoration recurred, usually in four days. Following the second and third courses, a progressive diminution of cough and expectoration took place with less tendency for recurrence. The coughing that had formerly manifested itself between arising and retiring was especially reduced. In four of the seven cases in this group, an elevated temperature became normal in a period of four to ten days. In three patients, temporary improvement took place, indicated by decrease in cough and expectoration, with recurrence of symptoms when the drug was terminated. In one far advanced patient, who had intestinal tuberculosis requiring an emergency operation for obstruction, the occurrence of severe nausea and vomiting prevented the administration of more than one course of the drug. This patient died four months after treatment was discontinued.

The case histories of three patients (1, 2 and 3) recited below illustrate the improvement that resulted from administration of para-aminosalicylic acid. The data of the entire group are shown in the accompanying table (Table II).

CASE REPORTS

Case 1: Male, age 37. *History:* Known tuberculosis since 1942. Previous treatment included bed rest, phrenic crush and gold therapy. During the six months before admission, the patient suffered from increasing weakness, shortness of breath and, during the last month, from fever and increasing cough. *Physical Examination:* Bronchovesicular breathing was heard in both lungs with moist rales in RLLF. X-ray film revealed far advanced bilateral disease, with three cavities in the left lung field and three in the right. The evening temperature prior to treatment reached 101.8 degrees F. *Course:* The patient was started on para-aminosalicylic acid at a dosage of 10 grams daily for a continuous period of three weeks, with a free interval of one week between courses. After the first seven days of treatment the temperature was normal and did not again rise above 99 degrees F. The cough was decreased, with reduction in sputum from 1 oz. daily to 1½ teaspoonfuls at the end of ten days. The red blood count rose from 3,100,000 to 4,500,000 in one month. The sedimentation

TABLE II

CLINICAL RESULTS OF PARA-AMINOSALICYLIC ACID IN CHRONIC PULMONARY TUBERCULOSIS

Case No.	Age	Sex	Extent and Nature of Disease	Dosage of Pas. Daily Grams	No. 3 wk. Courses	Clinical Results	Side Effects	Remarks
1.	37	M	Far-advanced multiple cavities, acute febrile exacerbation	10	3	Temp. fell from 101.8° to 99° in 7 days with reduction in cough and decrease in sputum from 1 oz. to 1 teaspoonful	No complaints	Gained 21 lbs. after 3 courses. Marked clinical improvement in seriously ill patient
2.	43	M	Bilateral disease with enlarged hilar glands, acute exudative spread to RLL	10.5	3	Temp. 103° dropped to 99°, after 3 days with reduction in cough	Appetite diminished	Marked clinical improvement with disappearance of exudative lesions on X-ray
3.	39	M	Extensive disease in left lung	10 to 12	4	Cough reduced 90%. Expectoration reduced 90%. Appetite improved	Nausea intermittently	Reduction in cough and expectoration with slight clearing of exudative lesions on X-ray
4.	26	F	Bilateral disease, apparently arrested, developed positive sputum	10	2	Expectoration was lessened and sputum retained negative on concentration and culture	No complaints	Decreased expectoration in patient. In good clinical condition
5.	52	M	Far-advanced disease on left. Intestinal tuberculosis.	6 to 8	2	Reduction in cough and expectoration	Severe nausea and vomiting	Nausea forced discontinuance of drug. Improvement temporary Previously, temporary improvement with streptomycin

6.	33	F	Far-advanced bilateral disease	5 to 10	2½	Cough reduced 70%, sputum reduced 90%	Severe nausea	Nausea forced discontinuance of PAS. Temporary improvement took place each course. Previously temporary improvement with streptomycin
7.	57	M	Disease advanced in left chest with large cavity	11	4	Cough and expectoration reduced 80% on each of the first three courses. Less effect on fourth. Temp. of 100° reduced to normal first 2 courses	Diminished appetite. 3 to 4 loose bowel movements daily	Improvement temporary during period of drug administration
8.	31	F	Advanced bilateral disease with cavity in LUL	10	1	Cough and expectoration reduced 80% after first week	Nausea and 3 loose bowel movements daily	Temporary improvement comparable to previous course of streptomycin
9.	32	F	Moderately advanced disease with cavity in LUL	10	2	Reduction in cough and expectoration	Diminished appetite	Reduction in cough in a patient in good clinical condition, with single cavity.
10.	42	M	Bilateral disease, apparently arrested, developed positive sputum.	10	1	Slight cough and expectoration almost disappeared in 4 days. Sputum retained negative after 1st course	Loss of appetite. 2 loose bowel movements daily	Reduction in cough in patient apparently convalescent and arrested
11.	53	M	Bilateral disease, fibro-productive	10	3	No effect	Decreased appetite. Loose bowel movements	Cough slight, not affected. No change in X-ray after 2 months
12.	37	M	Multiple cavities in both lung fields	10	1	Reduction in cough and expectoration in 4 days	Considerable nausea and vomiting	Could not continue drug beyond one course because of vomiting

rate showed no change, 26 mm. at the start of the course and 24 mm. at the end. The patient experienced a marked increase in general well being and a gradual gain in weight during the first course of treatment. At the end of four courses, i.e., four months, he had gained fifteen pounds in weight. During the fifth month the patient was given one gram of streptomycin daily for three weeks, with continued gradual gain in weight but without significant change in symptoms otherwise. The total weight gained was twenty-one pounds. X-ray film of the chest before and after each treatment showed a gradual clearing of exudative lesions, with decrease in the size of the large cavities in both lung fields and apparent disappearance of two small cavities in the right lung. No planographic x-ray examinations were made. During the period of administration of para-aminosalicylic acid, five determinations of the blood NPN and routine urine tests before and after treatment showed no changes.

The side effects of the drug were absent in this patient, who made no complaints whatsoever during its administration. Six months after treatment the patient had maintained a twenty-one pound weight gain. His dyspnea was improved, and he left the sanatorium for return to England. In this patient with far advanced active pulmonary tuberculosis, the acute symptoms of the disease were promptly controlled with conspicuous and marked clinical benefit. The persistence of multiple cavities in the lung indicate that no arrest of the disease was obtained, nor indeed was it expected in this type of case.

Case 2: Male, age 43 years. *History:* Three years before admission while the patient was in the Army, fatigue on slight exertion began. This was soon followed by intermittent fever as high as 104 degrees which became normal after one month's bedrest. There was no cough nor weight loss. X-ray filmo f the chest at this time revealed dense shadows near the hilum in the right lower lobe and the left midlung field. One examination of the gastric contents was positive for tubercle

CHART I

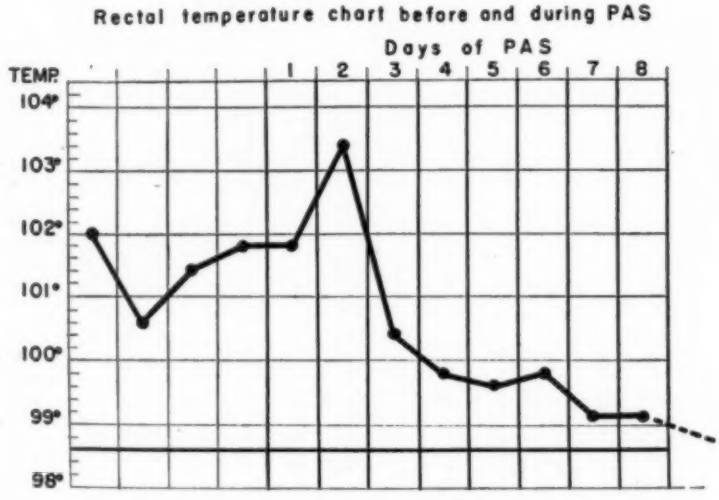

CHART 1: Prompt disappearance of fever after administration of Para-aminosalicylic Acid.

bacilli. He was discharged from the Army a year later, the lesions in the lung having shown little change by x-ray examination. The patient was ambulatory, and worked at a desk job. Two weeks before admission he suffered from cough, fatigue and increasing fever. *Physical Examination:* Bronchovesicular breathing and inconstant moist rales were heard over the right lower lobe. The fever ranged from 99 degrees F. to 103 degrees F. X-ray film of the chest revealed an exudative spread in the right lower lung field, in addition to the dense lesions previously observed. The sputum was negative on smear for tubercle bacilli, but subsequently proved positive on cultures. *Course: Because* of the unusual nature of the lesions penicillin was given intramuscularly for a period of eleven days without change in cough or fever. Para-aminosalicylic acid was begun with 10.5 grams being administered daily in five divided doses. The temperature promptly became normal in a period of three days, as shown in the accompanying chart (Chart 1). The cough disappeared on the fourth day of treatment and the patient revealed a marked increase in general well-being. After the first course of treatment, three weeks, the sedimentation rate decreased from 45 mm. to 29 mm. in one hour. X-ray film of the lungs showed a diminution of the exudative lesions in the right lower lung field, more marked after the fourth course as seen in Figure 1. Two additional courses of para-aminosalicylic acid were given. No changes were observed in the blood count, urine, or blood NPN after each course of therapy. The side effects of the drug in this patient consisted of a decrease in appetite, occasional nausea and a tendency to loose bowl movements. The latter was controlled by administration of one teaspoonful of metamucil morning and night. After a total of six courses of the drug, the patient stopped further treatment for six weeks. Recurrence of fatigue and cough took place. A chest x-ray film revealed an exudative spread at the base of the right

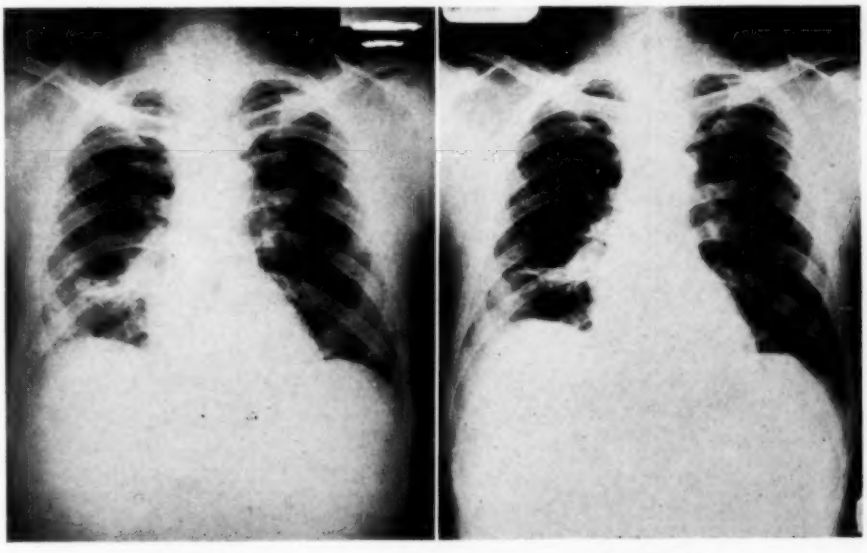

FIGURE 1 a FIGURE 1 b

Clearing of Exudative Spread after Four Courses of PAS.

Figure 1 a: X-ray before treatment.

Figure 1 b: X-ray after four courses of PAS.

lung. After three weeks of para-aminosalicylic acid, three grams twice daily, with increased rest, the exudative lesion again disappeared with loss of cough and fatigue.

The patient carried on his usual work as a design engineer during the first six months of treatment. Despite this activity a tuberculous exudative lesion was brought under prompt control during administration of PAS, recurring when the drug was stopped and again disappearing on treatment with PAS. Furthermore, a progressive decrease in what appeared to be hilar lymph node tuberculosis has taken place. At this time the patient is cooperating by accepting bed rest and administration of the drug.

Case 3: Male, age 39 years. *History:* Known history of tuberculosis since 1935, arrested in 1938 with reactivity in 1944. Since then the patient suffered from cough and expectoration, with weakness, anorexia and lassitude in year preceding treatment. *Physical Examination:* Entire left chest was dull to percussion. Bronchial breathing with moist rales over left upper lobe was heard. The right lung was clear. X-ray film showed the mediastinal contents pulled markedly to the left with dense fibrocavernous disease present in the upper lung field. *Course:* Five courses of PAS were administered, daily dosage 10 grams, each course consisting of three weeks with a free interval of one week. During the first ten days of treatment cough and expectoration were reduced 75 per cent. There was a considerable improvement in well-being, with increased appetite, despite occasional nausea. During the first two months, the x-ray film showed some clearing of exudative lesions in left lower lobe, but no change appeared after this time. The patient was referred to a hospital for thoracoplasty which was not thought advisable for him prior to treatment. Whereas the patient was formerly constipated, he had one normal movement daily on administration of PAS. No effect was noted on the blood NPN, blood counts or urine as a result of administration of the drug.

Discussion

Although the series of cases was small, decrease in cough and expectoration was consistently observed. Clearing in exudative lesions was noted in three patients, as well as a reduction in elevated temperature to normal. No significant change in the size of the cavities of these patients took place, except in one patient in whom it appeared that two small cavities disappeared during the administration of five courses of the drug.

The reduction in cough and expectoration was especially noted during the day, occurring in a period of less than one week, with gradual and sustained reduction in symptoms in three cases. A feeling of well-being accompanied reduction in cough and expectoration, even in cases in which loss of appetite and intermittent nausea took place. The side effects of the drug necessitated discontinuation of treatment in three patients. No serious toxic effects were encountered.

The most consistent side effect of the drug was diminution

in appetite, which took place in all except two patients. In two others nausea and vomiting were severe enough to cause cessation of the treatment. In six of the twelve patients, two or four loose bowel movements occurred daily, which was counteracted to a considerable degree by the administration of metamucil. In nine patients, determination of the blood NPN was carried out. An elevation was noted in one case with far advanced intestinal tuberculosis, which returned to normal in two weeks. Albumin was noted in the urine in two patients, which disappeared after discontinuing the drug for a few days and did not return when the drug was again administered. Five patients who had an elevated erythrocyte sedimentation rate showed a reduction after the first course of treatment.

Para-aminosalicylic acid would appear to be a therapeutically useful drug in patients with pulmonary tuberculosis, especially those in whom an acute exacerbation of chronic disease has occurred and in whom streptomycin is withheld because of the fear of development of resistance to the latter drug. Despite the statement of Lehmann that resistance does not take place as a result of continued para-aminosalicylic acid therapy, our observations of the concentration of the drug required to inhibit the growth of tubercle bacilli after treatment in three cases suggests that additional investigation is desirable.

Further experience with the drug may result in its administration with fewer side effects. Clinical benefit has recently been observed with a smaller dosage, such as six to nine gms. daily. Consideration should also be given to the possibility that smaller amounts of the drug, such as six or eight gms. per day, may be useful when combined with other antibiotics, such as promizole and streptomycin.

SUMMARY

The serum of patients and normal individuals after ingestion of 2 to 4 gms. of para-aminosalicylic acid produces a definite inhibition of the growth of the tubercle bacillus in Dubos media. This has been demonstrated with the H37Rv strain as well as with strains recovered from patients with active pulmonary tuberculosis.

Para-aminosalicylic acid was administered to twelve patients with advanced chronic pulmonary tuberculosis. The dosage employed was 10 to 11 gms. daily in courses of three weeks, with a free interval of one week. In seven patients in whom the symptoms of active infection were sufficiently marked to permit judgment of the effectiveness of drug, observable clinical improvement took place in all. In three patients recurrence of symptoms on cessa-

tion of treatment took place. Three patients who suffered from fever and signs of acute exudative tuberculosis were much improved and in good general condition after treatment, but without arrest of their disease.

The characteristic effects of administration of para-aminosalicylic acid were prompt reduction in cough and expectoration, and decrease in fever when present. These favorable results were generally manifested within the first week of treatment. The side effects of the drug were intermittent nausea, vomiting and looseness of the bowels. In three of the twelve patients these symptoms were sufficiently severe to force discontinuance of the drug.

The favorable clinical results of treatment with para-aminosalicylic acid in chronic pulmonary tuberculosis suggests that this drug is a useful agent in the treatment of acute exacerbation of the disease.

The possibility that administration of para-aminosalicylic acid with other antibiotics, such as promizole and streptomycin, would appear to deserve investigation.

Since this paper was written, PAS has been administered in one or more courses to ten additional patients with chronic pulmonary tuberculosis. Clinical improvement took place in all instances, characterized especially by reduction in cough and expectoration and improvement in well-being. Side effects were few on a dosage of 3 gms. three times daily. A favorable clinical response was also noted in patients who had become resistant to streptomycin. In the treatment of cavitary pulmonary tuberculosis by lung immobilizing therapy in the equalizing pressure chamber, closure of cavity has taken place in a number of cases of advanced and moderately advanced tuberculosis;[14] in a few cases recently treated by this method PAS has been added in the hope of shortening the time required for cavity closure.

RESUMEN

El suero de pacientes y de individuos normales que han ingerido de 2 a 4 gramos de ácido para-aminosalicílico produce una inhibición bien definida del crecimiento del bacilo tuberculoso en el medio de Dubos. Se ha demostrado esto con la cepa H37Rv y tambien con cepas obtenidas de pacientes con tuberculosis pulmonar activa.

Se administró el ácido para-aminosalicílico a doce pacientes con tuberculosis pulmonar crónica avanzada. La dosis empleada fue de 10 a 11 gramos diarios en series de tres semanas, con un intervalo libre de una semana.

En siete pacientes en los que los síntomas de infección activa fueron lo suficiente marcados para permitir que se formara un juicio acerca de la eficacia de la droga, tuvo lugar mejoría clínica

observable en todos ellos. En tres pacientes tuvo lugar recrudecimiento de los síntomas al pararse el tratamiento. Tres pacientes que tenían fiebre y signos de tuberculosis exudativa aguda quedaron muy mejorados y en buena condición general después del tratamiento, pero sin estacionárseles la enfermedad.

Los efectos característicos de la administración del ácido para-aminosalicílico fueron la pronta reducción de la tos y la expectoración y la disminución de fiebre cuando existía. Por lo general se manifestaron estos resultados favorables durante la primera semana del tratamiento. Los efectos laterales de la droga fueron: náusea intermitente, vómitos y diarrea. En tres de los doce pacientes fueron estos síntomas lo suficiente severos para obligar que se descontinuara la droga.

El favorable resultado clínico del tratamiento de la tuberculosis pulmonar crónica con el ácido para-aminosalicílico sugiere que esta droga podrá ser un agente útil en el tratamiento de las exacerbaciones agudas de la enfermedad.

La posibilidad de administrar el ácido para-aminosalicílico con otros antibióticos, tales como el promizol y la estreptomicina, parecería merecer investigación.

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Streptomycin in Tuberculosis, Clinical Experience*

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Summary†

1) Seventy-seven cases of tuberculosis have been treated, presenting various clinical pictures with varying complications and foci of localization. Of the total, 66 cases have been analyzed with the following results: 33 have finished the treatment, 17 are continuing, 8 have been discontinued, and 5 have died.

2) All of the patients were under treatment in the sanatorium. The diagnosis was substantiated in each case by finding tubercle bacilli in the respective specimens. Sixty-six per cent of all patients who completed the treatment have gained weight, some as much as 18 Kg. Favorable changes have been noted in the temperature curves, and in some cases the curve has returned to its original contour regardless of its initial shape. Tuberculin skin sensitivity, which was negative at the onset in 5 cases, turned positive during the 30 days of treatment.

Bacteriologic studies showed a marked decrease in the excretion of bacilli, and conversion occurred in 15 cases. The changes in the sedimentation rate were consistent with the clinical improvement. In almost all cases, regardless of the final outcome, marked detoxification was observed, with remission of functional symptoms, improvement of appetite, and typical euphoria.

3) Aside from its favorable effect on recent exudative lesions, streptomycin is offered as a form of chemotherapy for tuberculous exacerbations.

4) In conjunction with accepted rational therapy and insulin, it has proved effective in exudative flare-ups of pulmonary tuberculosis in diabetics.

5) In the treatment of pulmonary tuberculosis in pregnancy, streptomycin has proved effective. Pregnancies have been carried to term successfully with normal deliveries and with evident improvement in the pulmonary lesion of the mother. The dosage in

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these cases should be moderate, and toxic reactions in the mother do not necessarily warrant discontinuation of the treatment. It seems that the greater resistance of the fetus, or its greater tolerance for the drug, are due to the higher alkalinity of the fetal blood.

6) In the early stages of tuberculous empyema when the pleurae have not been appreciably damaged, it is permissible to use the intramuscular route alone with conventional drainage procedures performed at the proper time. In more advanced cases of frank empyema, one should employ proper drainage and obliteration of the pleural cavity by phrenicotomy or thoracoplasty in conjunction with streptomycin.

7) In cases of recent dissemination treatment should be started as early as possible. Persistence of the radiologic findings with an improvement in the clinical picture should be sufficient indication for institution of proper collapse therapy according to the case.

8) Cases of tuberculous laryngitis and tracheobronchitis derive considerable benefit from streptomycin. The intramuscular route is preferred in these cases. Aerosols have not proved as effective as was anticipated; hence, if employed, should always be used in conjunction with intramuscular administration. Apparently complete cures of tuberculous laryngitis have been obtained in cases of hematogenous tuberculosis.

9) Acute, subacute, and chronic hematogenous pulmonary tuberculosis and their extrapulmonary localizations are definite indication for streptomycin therapy which should be started without delay as soon as this diagnosis has been established. Improvement of lesions associated with hematogenous pulmonary tuberculosis has been observed in the following foci: testes, joints, pleurae, kidneys, and superficial lymph nodes.

10) In cases of tuberculous enteritis associated with various types of the disease, treatment has been successful in alleviating all the symptoms, and in some cases even regression of the radiologic picture has been noted. In others, radiologic evidence of cicatricial involvement is still present. As yet, we cannot state that healing of the enteric lesions is definite until a longer follow-up study of the patients has been carried out.

11) Twenty-eight of the patients have shown no reactions to the drug. Eight had local reactions, and 12 had reactions of an allergic nature. Of these 12, seven had appreciable febrile reactions, three had urticaria, one a morbilliform exanthema and one a maculopapular exanthem. This latter presented a problem of considerable importance because the reaction produced an exanthem and the patient could not be fed properly. In addition, this patient had hemorrhagic infiltrations in the palms. In seven cases

the drug produced nausea and vomiting. Two showed alterations of the renal function in the form of oliguria. The complete vestibular syndrome has not been observed in any case, but fourteen had headaches with or without nausea and vomiting, and five have complained of dizziness and mild vertigo. As for hearing capacity, three have had tinnitus with slight hypacusia, and one had transient hyperacusis. One case had marked somnolence and another had joint pains.

The best results have been obtained with the following indications:

(1) Any superficial mucosal lesion in the mouth, larynx, bronchi, or intestines.

(2) Any localized lympho-glandular lesion, especially when there is spontaneous drainage.

(3) Any case of hematogenous dissemination, especially pulmonary, but also less effectively, of extrapulmonary origin.

(4) In the lung, any perifocal reaction, or any specific recent exudative reaction, regardless of the route of dissemination.

This summary is presented as evidence of the interest with which we have used and studied streptomycin, and is offered as our modest contribution to the voluminous statistical data of American clinicians.

Extrapleural Pneumonolysis with Plombage Versus Thoracoplasty

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Thoracoplasty has always been one of our most reliable operative procedures in the surgical treatment of pulmonary tuberculosis and although the disadvantages of this operation are numerous, it still yields the highest percentage of sputum conversions and cavity closures. Throughout the surgical literature the procedure of extrapleural pneumonolysis with various "plombe" materials[1] is encountered as a rival to thoracoplasty and its exponents compare its capabilities as a collapse procedure with this latter operation. Certainly it obviates the objectionable features of thoracoplasty and from a theoretical standpoint should produce as satisfactory an end-result.

It is interesting to observe how optimistic many authors were regarding the results obtained with the extrapleural operation as employed in selected cases.[2-12] In the following tabulation the various advantages for extrapleural pneumonolysis with plombage over thoracoplasty are enumerated with notations as to the various exponents of each advantage:

1) Expectoration made easier preventing aspiration pneumonia;[2,4,9,10,13,14]
2) Cosmetic effect—no deformity;[4,10]
3) Selective collapse with maximum preservation of normal pulmonary parenchyma and respiratory function;[4,9,15-18]
4) Abnormal conditions for heart and lungs not produced as with thoracoplasty;[2,4]
5) Reduced possibility of paradoxical respiration as seen in thoracoplasty;[4,9]
6) Bilateral lesions are no contraindication to it;[9-11,13,15-17,19]
7) Only one operation is necessary;[10]
8) Thorax maintains its function;[2,18]
9) Results are immediate.[4]

Although many early authors praised extrapleural pneumon-

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*From the Department of Medicine and Surgery, Veterans Administration, Oteen, North Carolina; published with permission of the Chief Medical Officer, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

olysis regardless of the type of pack employed, the majority made the reservation that extrapleural packing should be used only in selected cases, and that it was inferior to thoracoplasty. Baer[4,5] stated that pneumonolysis with pack was a worthy substitute for thoracoplasty in selected cases, and deWinter and Sebrechts[6] and Winternitz[15] actually considered the operation superior to thoracoplasty when indications were closely adhered to, and substituted it for thoracoplasty in such cases. Aufses[16] in 1929 stated that extrapleural pneumonolysis was never indicated as a substitute for thoracoplasty and that up to that time no known method of extrapleural stripping with plombage had withstood careful investigation. He summarized his article by affirming that the procedure of extrapleural pneumonolysis is useful in only an occasional case of pulmonary tuberculosis and that the percentage of failures was so high that he questioned the usefulness of the operation.

We agree with Aufses so far as the usefulness of the operation is concerned, and our results have clearly demonstrated that the procedure is never superior to thoracoplasty. As to its use in selected cases, or in cases with precise indications where thoracoplasty is contraindicated, we feel that the majority of patients would be far better off without any major collapse procedure rather than undergo an extrapleural pneumonolysis with any form of plombe material including lucite, air, or gomenol.

In the past two years we have performed 14 operations of extrapleural pneumonolysis with lucite plombage as primary major collapse procedures. In each of these cases indications for the procedure were closely followed and were considered optimum for the procedure. Of these, four patients, or 27.1 per cent, converted their sputum. Eight, or 57.1 per cent of the lucite packs have had to be removed because of complicating factors. Fortunately the mortality rate has been zero.

During the past five years our department has performed 1,128 stages of thoracoplasty upon 415 patients. The mortality rate has averaged 0.75 per cent per operation. The sputum conversion rate with the far advanced, often bilateral type of lesion which we accepted for operation averaged 65 per cent.

These figures show that our percentage of sputum conversions and cavity closures has been low in the patients treated with extrapleural pneumonolysis with lucite pack, and complications have been numerous. The procedure has been abandoned on our surgical service and it is felt that all of the lucite packs still *in situ will* have to be removed during the next few months. A comparison of these results with those obtained through thoracoplastic procedures demonstrates that thoracoplasty still remains our most reliable collapse method of treatment.

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Pneumoperitoneum Therapy in Lower Zone Tuberculosis

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The treatment of pulmonary tuberculosis has been and is a complex problem. In general, rest has been advocated as a beneficial measure in cases with clinical or radiological activity. Unfortunately, rest alone is not usually effective except in limited disease without cavitation. As the lesion of pulmonary tuberculosis becomes established, the picture becomes complicated. Adhesions of the pleura often are responsible for mechanical limitation of the respiratory function of the involved lung. Furthermore, fibrosis is a very frequent major complication producing distortion of the mediastinum and its contents, also fibrosis may be diffuse upsetting the normal physiology of gaseous exchange through the pulmonary alveoli. Many cases in responseto immunological phenomena peculiar to infection with tubercle bacilli show either localized or extensive caseation.

Before one attempts to outline a course of treatment for a case of pulmonary tuberculosis, an intensive investigation of the patients as a whole is desirable. Let us for simplicity's sake consider that we are dealing with a group of patients whose only problem is that of pulmonary tuberculosis. We must then, first of all, get a picture of the activity of the case. Are the lesions progressing or regressing? To what extent has pulmonary fibrosis progressed? If there is extensive fibrosis, is there adequate absorption of oxygen and elimination of CO_2?

One must also attempt to evaluate the degree to which the pleura has been involved. Here the history of pre-existing pleurisy is important. Further studies might include bronchospirometry. In those cases in which the pleura is radiologically thickened the problem is straightforward. It is necessary definitely to determine the location, size and character of pulmonary cavities which may be present. Peripherally placed lesions collapse with greater regularity than those more medially situated. Is it an atmospheric, positive, or negative pressure type of cavity? Are the walls of the cavity hard, or soft, is the cavity situated in soft collapsable lung, or is there extensive adjacent fibrosis? A further consideration is that lower zone cavities in tuberculosis are frequently in the apex of the lower lobe. This location is of course

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central. As has already been stressed relaxation of centrally placed pulmonary tissue is not maximum.

There are several mechanical methods of treatment for pulmonary tuberculosis with cavitation advocated to date. In 1931 Dr. Banyai made a new step in the therapy of pulmonary tuberculosis. He used and widely advocated the introduction of air into the peritoneal space. As is well known the intrapleural pressure is negative. The pressure in the peritoneal space is neutral, except in the subdiaphragmatic region where it is negative. Suspended between these two pressures is the diaphragm. It is obvious then that to increase the abdominal pressure would favor an elevation of the diaphragm. Obviously a point against this therapy is that there is bilateral elevation of the diaphragm. In an attempt to give more selective collapse, numerous workers have advocated phrenic interruption on the desired side. This has been very effective, often increasing the elevation of the involved diaphragm by 2 to 8 cm. Since the original paper of Dr. Banyai, many articles have been published and it is the opinion of most workers that lower and mid zone lesions have responded more favorably than those higher placed.

Here then is a form of therapy which appears peculiarly suited to disease involving the lower and mid zone of the lung. The therapy of lower zone tuberculosis is generally considered far from satisfactory. As we have already mentioned, thoracoplasty is best suited to upper zone disease. A limited lower thoracoplasty is technically an unsatisfactory procedure. To use the orthodox thoracoplasty which collapses the lung from above downwards is a wasteful procedure in that healthy lung is unnecessarily defunctionalized. If the lesion is high in the lung, pneumothorax is often beneficial but here again considerable good lung is put out of order in an attempt to effectively collapse the lower zone. Also, pneumothorax is far from innocuous. All who have used this procedure know the frequency of adhesions which require division, this is often impossible and in the others pleural effusion and empyema are by no means uncommon. Even in the technically successful pneumothorax we are often left with a crippled lung due to pleural thickening.

On the other hand, pneumoperitoneum is a relatively safe and easy procedure. There is no absolute location at which this must be done. It seems however that it should not be done over the liver as it has been shown that here puncture of the liver may easily be the cause of an air embolism. Also it is doubtful whether the right lower abdominal quadrant should be used, as here the possibility of adhesions from appendiceal inflammation is obvious. It has been mentioned that puncture of the bowel may com-

plicate pneumoperitoneum. This has been very rare. However, if the bowel were adherent to the peritoneum at the site of induction, it most likely would occur. Thus most workers induce pneumoperitoneum on the left side. Initially 400-700 cc. of air are given. The amount is largely determined by the size of the patient and the objective symptoms which the patient presents. If too much air is given there is usually referred diaphragmatic pain in the shoulders. In maintaining the pneumoperitoneum, air is given periodically at approximately weekly intervals, usually 800-1000 cc. Here again the patient's comfort is the chief point in fixing the quantity to be given. Usually in a short period of time the patients are asymptomatic following pneumoperitoneum. Occasionally individuals complain of anorexia, this is thought to be due to dislocation of the stomach and bowel. Anorexia can usually be obviated by proceeding with therapy in a cautious manner. Peritoneal adhesions are occasionally seen, but these do not appear to affect either the treatment or the effectiveness of the procedure. A few cases have been observed where peritoneal effusion has followed pneumoperitoneum. This has not appeared to effect the patients progress and is usually not considered to contraindicate continuation of therapy. No doubt, peritoneal effusion occurs in those who have tuberculous involvement of their peritoneum. It is interesting to note here that numerous workers feel that air injection into the peritoneal cavity is beneficial in cases of tuberculous peritonitis. A point which seems important to us is that there is little possibility that we will produce a crippled lung as so frequently occurs in pneumothorax because here the pleura is not affected.

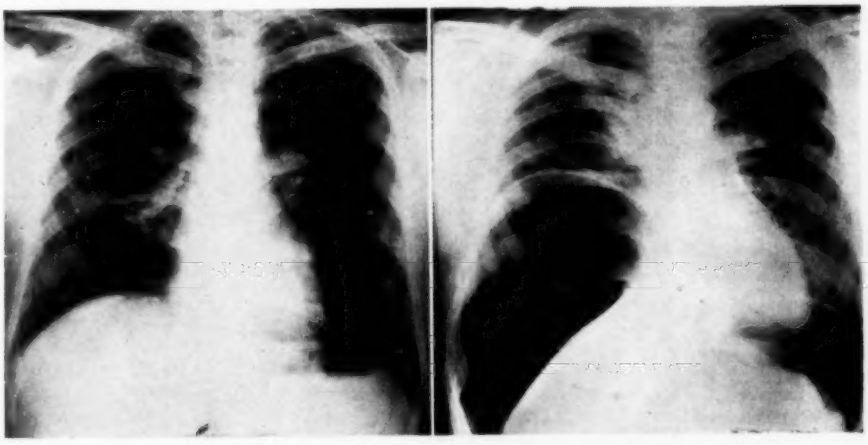

FIGURE 1 FIGURE 2

Fig. 1: There are abnormal shadows in the right lower chest with cavitation laterally and below the right hilus.—*Fig. 2:* A phrenic crush has been done on the right side, giving more selective collapse on this side.

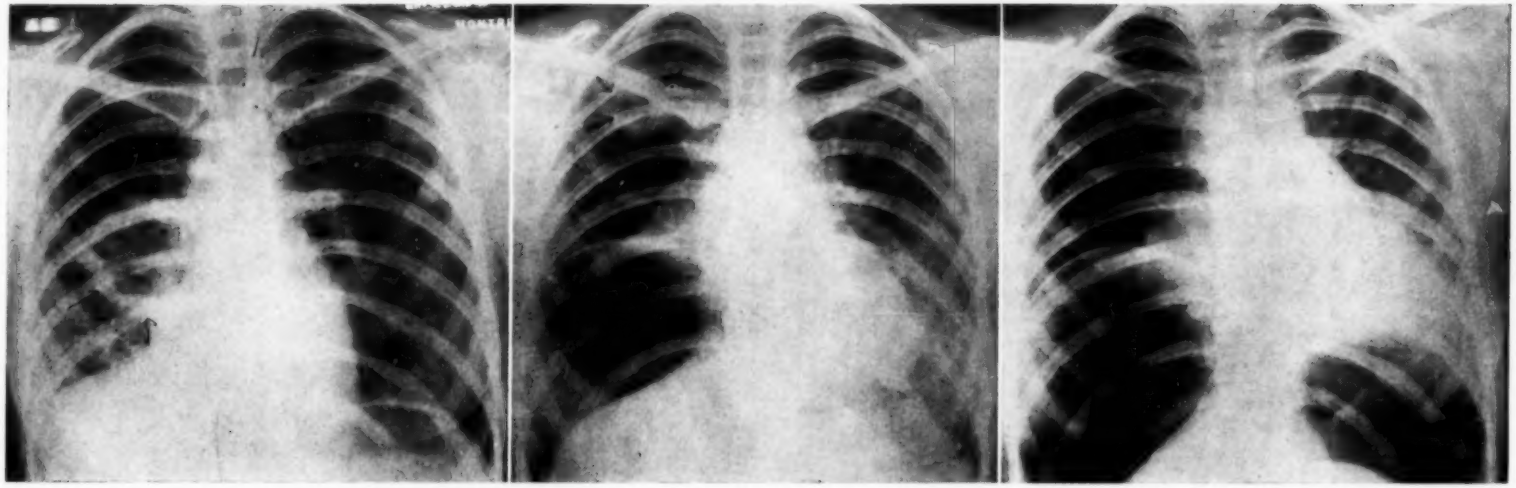

FIGURE 3 FIGURE 4 FIGURE 5

Fig. 3: There are abnormal shadows in the right base with a large cavity just below and lateral to the right hilus. On the left side, there are abnormal shadows in the 2nd interspace anterioly laterally placed.—*Fig. 4:* Pneumoperitoneum has been induced. There is a good rise of the right diaphragm associated by a phrenic crush right. There is a new area of parenchial disease in the right upper zone complicated by cavitation. There is regression of the disease on the left side.—*Fig. 5:* Pneumothorax is induced on the right side with obliteration of the upper zone cavity. Pneumoperitoneum is being maintained. Pneumothorax on the right side has been abandoned. The lesion on the left side becoming more fibrotic in nature.

CASE REPORTS

D. P. was admitted to the hospital on June 15, 1946. The duration of disease was indefinite. He was in good physical condition, afebrile and his pulse was regular and normal. Examination of the chest revealed only dullness over the right base posteriorly. Radiologically there was moderately advanced exudative pulmonary tuberculosis, involving the right lower lobe. There were two small cavities at the apex of the right lower lobe, visualized most clearly by tomographic studies (Fig. 1).

In this case it was felt that pneumoperitoneum offered the best chance of arrest of the disease. Pneumoperitoneum was started in July 1946 and a right phrenic crush was done on July 25, 1946. His sputum decreased in amount from 1 ounce to drachms 2 daily. It is interesting to note that while in this hospital no positive sputums were obtained on this patient, prior to induction of pneumoperitoneum. However, sputum examination done a month prior to admission showed that his sputum was positive for tubercle bacilli. Following induction of pneumoperitoneum, there has been good relaxation of the lower half of the right lung (Fig. 2). His sputum has remained negative to date, eight months after establishment of treatment.

Patient's clinical condition has remained satisfactory. His weight has been maintained. He is afebrile, pulse continues normal. Haematological studies indicate that there has been diminished activity, there being now a low sedimentation rate and the white blood count only slightly above normal.

M. I. was admitted to this hospital on August 1, 1944. On admission patient was found to be underweight. Her blood pressure was normal. Her abdominal examination showed no abnormality. Her genito-urinary system appeared to be within normal limits. Examination of her chest showed bilateral pulmonary tuberculosis with a giant cavity in the right lower lobe and soft parenchymal lesions in the left apex and left infraclavicular region (Fig. 3). On admission, sputum was Gaffky 5. In this case pneumoperitoneum was done and this was made more selective by doing a phrenic crush on the right side on October 13, 1944 (Fig. 4).

Shortly after the institution of this treatment, the cavity in the right lower lung diminished in size and over the succeeding four months the cavity gradually disappeared, being no longer demonstrable either clinically or radiologically. Coincident with the radiological disappearance of the cavity in the right lower zone, sputum became negative for tubercle bacilli.

In August 1945 it was noted that there was cavitation in the right apex and effectively to collapse this area, a pneumothorax was induced on the right side in November 1945 (Fig. 5). The collapse was satisfactory and there was obliteration of the cavity in the right upper zone. Progress in this case has been good. Temperature has remained afebrile and sputum has been consistently negative since the latter part of 1944. In this case atelectasis of the right lower lobe developed as a complication shortly after the initiation of treatment. This atelectatic lobe was observed, and it was found that a minimal degree of bronchiectasis developed, but this was not a complicating factor in the disease, as there was no excessive expectoration of sputum and there were no signs of infective episodes. Bronchoscopy revealed an apparently normal bronchial tree, except for some submucous granulation in the right lower lobe bronchus proper.

FIGURE 6

FIGURE 7

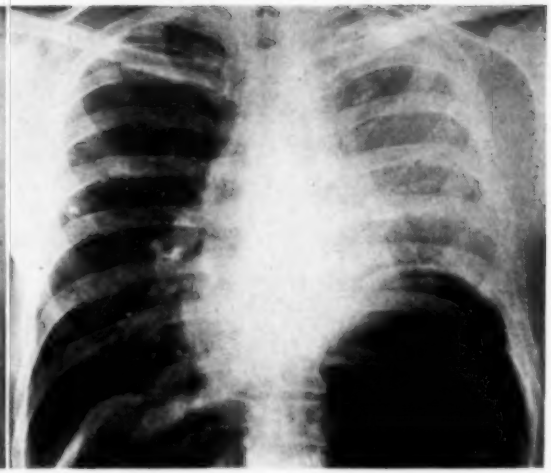

FIGURE 8

Fig. 6: There is a pneumothorax present with approximately 50 per cent collapse. Pneumothorax is complicated by an adhesion in the upper part. There is a large inflated cavity in the mid zone of the collapsed lung.—*Fig. 7:* The adhesion present in figure one has been divided. There is now approximately 75 per cent collapse of the left lung. The cavity is still present but considerably reduced in size.—*Fig. 8:* Pneumothorax has been abandoned. There is a considerable rise of both halves of diaphragm. The left diaphragm is considerably higher than the right, due to the phrenic crush on the left. The left lung is partially obscured by pleural reaction. The cavity however is not visualized.

During the period of subsequent observation, her sputum has been persistently negative and there has been progressive resolution of the densities in both lungs.

J. C. was admitted December 4, 1944. On admission patient showed a moderate degree of wasting. Temperature was normal. There were no signs of physical abnormality, other than in examination of the chest which showed dullness at the left apex posteriorly, with scattered rales in this area. There were signs of a pneumothorax on the left side. X-ray film of the chest showed a 50 per cent collapse of the left lung by pneumothorax. Pneumothorax was inefficient due to an adhesion and there was a large tension cavity present in the upper half of the left lung (Fig. 6). Bronchoscopy, January 1945, revealed tuberculous bronchitis.

In November 1944 a pneumonolysis was done on the left side and there was a good collapse of the left lung (Fig. 7), however, the cavity was still present, although somewhat diminished in size. Shortly after pneumonolysis, patient developed extensive pleural effusion on the left side and it was thought inadvisable to continue pneumothorax. Pneumothorax was allowed to re-expand and the fluid was aspirated from the left chest. In May 1945 a phrenic interruption was carried out on the left side and this was followed by the induction of pneumoperitoneum in July 1945. There was almost immediate obliteration of the cavity present in the left upper lung (Fig. 8). Patient has been observed from this date to March 1947 and has shown a persistent normal temperature with normal pulse. Weight has increased 30 pounds. Haematologically, there are no signs of activity. Sputum has remained consistently negative since May 1945.

G. F. was first diagnosed as suffering from pulmonary tuberculosis in 1941 in a Tuberculosis Clinic in this city. Bed rest was carried out for seventeen months at home without any appreciable improvement in the pulmonary condition. In April 1946 patient was admitted to hospital for further treatment.

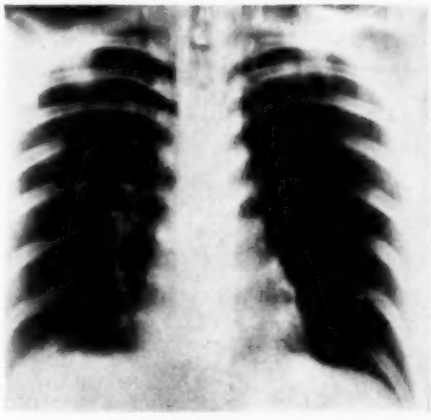

FIGURE 9

FIGURE 10

Fig. 9: Tomogram 6 cm. from the skin of the back, shows a large cavity in the right hilar region.—*Fig. 10:* Pneumoperitoneum has been established and a phrenic crush has been done on the right. Note the rise of both halves of the diaphragm, greater on the right. There is good relaxation of diseased area. There are also lesions in the upper portion of the left lung.

Physical examination showed a well developed, well nourished girl with normal temperature and normal pulse. There was no sputum and gastric lavage was negative. The examination of the lungs showed a clear left lung. On the right side there were moist rales and bronchial type of breathing over the upper portion of the right chest. Radiologically, there were signs of soft parenchymal lesions above the 2nd rib anteriorly on the left, and near the right hilus there was a large cavity with soft parenchymal lesions throughout the right lower lobe (Fig. 9). In spite of the negative sputum on admission there was no doubt as to the etiology of this disease, as the patient had previously been proven to have positive sputum. Consequently, an attempt was made to obliterate the cavity in the right hilar region by pneumoperitoneum and a phrenic crush on the right side, this treatment being initiated in May 1946. There was a very satisfactory rise of both halves of the diaphragm, greatest on the right, and there has been radiological disappearance of the cavity in the right hilar region (Fig. 10). The lesion in the left apex has remained stationary.

Clinical progress has been good. Temperature ran a low subfebrile course until January 1947, ten months after institution of collapse therapy. Temperature, however, has been normal for the last three months. Patient has no sputum and repeated gastric lavages have been negative both by smear and culture. There has been persistent obliteration of the cavity in the right hilar region. Weight is being maintained satisfactorily.

CONCLUSIONS

1) Pneumoperitoneum is a safe and simple procedure.

2) Excellent collapse of the lower 2/3 of the lung can be achieved by this means.

3) An added phrenic nerve interruption gives more selective collapse of the diseased side.

4) Duration of pneumoperitoneum treatment is an unestablished factor, but it seems that it should be maintained as long as one would a pneumothorax.

5) Cases are presented which demonstrate the effectiveness of this therapy.

CONCLUSIONES

1) El neumoperitoneo es un procedimiento salvo y sencillo.

2) Por este medio se puede obtener un colapso excelente de las dos terceras partes inferiores del pulmón.

3) La adición de la interrupción del nervio frénico da un colapso más selectivo del lado afectado.

4) No se ha establecido todavía la duración de la neumoperitoneoterapia, pero parece que se deberia mantener por un tiempo igual al que uno mantendría un neumotórax.

5) Se presentan casos que demuestran la eficacia de esta terapia.

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Carcinoma of the Lung with Nonmalignant Pleural Effusion: Recovery by Pneumonectomy*

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One of the universally accepted contraindications to operability of a primary bronchiogenic carcinoma of the lung is the presence of metastatic pleural effusion. Such effusions occur in approximately 11 to 15 per cent of all bronchiogenic carcinomata.

Diagnosis of metastasis to the pleura is made by the finding of tumor cells in the fluid. The usual method for the study of tumor cells is that of Mandelbaum,[1] in which the fluid is centrifuged down and a sediment obtained, which, after hardening with formalin, is treated as ordinary tissue and stained with hematoxylin and eosin. The addition of glacial acetic acid to the fluid to bring the acid to 2 per cent is recommended by Foot[2] in order to prevent coagulation. By the Mandelbaum method, approximately 60 per cent of pleural effusions due to malignancy will show tumor cells. The remaining 40 per cent are missed for the following reasons: (1) Single tumor cells may be difficult to differentiate from the cells of long standing transudates. (2) The tumor may not shed cells. (3) The fluid is a transudate due to venous obstruction by a tumor mass. When, therefore, tumor cells are not seen in effusions associated with carcinoma of the lung, it is logical to assume that the fluid is in the 40 per cent group and that the patient therefore has a metastatic effusion. As a result, it has been generally accepted that the presence of any fluid whether it is serous or bloody is a contraindication to operation.

That pleural effusion due to causes other than malignancy may occur in patients with bronchogenic carcinoma is exemplified in the following case:

V.D., age 50. Entered the Jewish Hospital on June 30, 1943 and was discharged on August 23, 1943. The patient had a gradual onset of a chronic productive cough starting 1½ years ago. The cough became worse about six months prior to admission to the hospital at which time he began to lose weight. One week prior to admission he began to have chills, fever and dyspnea.

Physical Examination: Temperature 101 degrees F.; pulse 76; respiration 24 per minute; blood pressure 108/68. The patient was a thin emac-

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iated white male who looked chronically ill. He was well oriented. The important physical findings were diminished expansion of his left chest, diminished tactile fremitus, dullness to flatness posteriorly from the angle of the scapula downward. Breath sounds were vesicular elsewhere. In the right base there was a moderate number of coarse rales.

Laboratory Findings: RBC 3,510,000; WBC 13,000; Hgb. 69 per cent. Differential blood count, normal. Urine, negative. Sputum, purulent; showed no pneumococci, fungi or acid-fast bacilli. Roentgenogram of the chest revealed a shadow obliterating the left costophrenic angle, interpreted as pleural effusion.

From the left pleural space 1500 cc. of sero-sanguinous fluid was removed with considerable improvement of the patient's dyspnea. The fluid had a specific gravity of 1018, was sterile on culture, showed no bacteria on direct smear and a section sediment showed numerous polymorphonuclears and solid-staining cells with eccentrically placed nuclei. No tumor cells were seen. Another aspiration obtained 400 cc. of a similar fluid and again the sediment showed no tumor cells. Fluoroscopy following the thoracentesis revealed collapse of the left lower lobe previously hidden by the fluid. Bronchograms showed obstruction of the

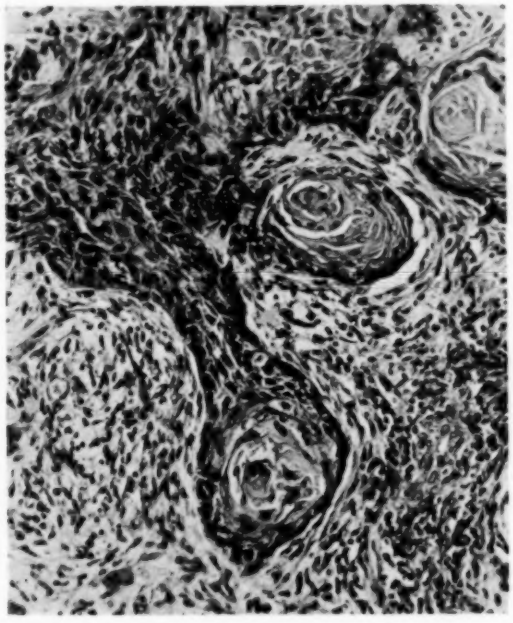

FIGURE 1: Bronchoscopic biopsy of left lower lobe showing squamous cell carcinoma.

left lower lobe bronchus and bronchiectasis of the right lower lobe. Bronchoscopy revealed the left lower lobe bronchus completely obstructed by a hard cauliflower growth. Section showed a squamous cell carcinoma (Fig. 1). The patient was given a course of sulfadiazine which apparently lowered his fever and on July 13, 1943 a left pneumonectomy was performed by Dr. J. L. Mudd. The pleural cavity showed no evidence of metastasis. The lung showed atelectasis of the left lower lobe. On opening the bronchus, a mass could be seen obstructing the lumen and extending down the bronchus for a considerable distance. The bronchi

beyond the point of obstruction were dilated and contained a large amount of yellowish pus. On cut section, the lung tissue appeared quite compact and contained numerous small abscesses.

Microscopic section through the tumor mass showed erosion and replacement of the bronchus by a group of large polygonal cells, varying in size but fairly well differentiated. The tumor had grown in small sheets, some of which were necrotic in the center. There was invasion of the underlying lung with marked infection and fibrosis. The pleural surface of the lung showed no tumor invasion. The alveoli throughout showed large phagocytic cells. The left upper lobe was negative. No evidence of carcinoma was seen in the tracheo-bronchial nodes. Section through an additional resection of the bronchus following pneumonectomy revealed no evidence of malignancy.

One month following the operation, the patient developed empyema as a result of the bronchial stump opening. Thoracotomy was performed and because of the persistent drainage, thorocoplasty was done and completed in January of 1944. He had one severe episode of a pneumonitis of the right lower lobe, apparently associated with his right lower bronchiectasis which was present prior to the operation. This cleared up promptly following bronchoscopy. In August, 1944 a diagnostic bronchoscopy was done and the entire tracheobronchial tree was clean. A recent examination showed no evidence of recurrence of the malignancy. He was well five years after operation. The only symptoms are moderate cough and sputum due to the bronchiectasis of the right lower lobe bronchi.

The pleural effusion in this patient was an inflammatory one. It is well known that hemorrhagic effusions may be due to infection with pyogenic micro-organisms. In Berliner's[6] series, 65 per cent of hemorrhagic effusions were due to malignancy and 11 per cent to inflammations of various types. It is obvious that a patient with bronchiogenic carcinoma may develop pleural effusion due to secondary atelectasis and infection, and that this effusion is nonmalignant.

SUMMARY

A 50 year old man with proved bronchiogenic carcinoma of the lung had a large hemorrhagic pleural effusion. Study of the fluid showed no tumor cells, but evidence of inflammation. A pneumonectomy was performed and the fluid was shown to be due to secondary pneumonitis. The patient is well after five years postoperatively.

The presence of pleural effusion in cases of bronchiogenic carcinoma of the lung does not contraindicate operation unless there is unmistakable evidence of metastasis to the pleura such as tumor cells in the fluid or visible tumor in the pleura.

RESUMEN

Un hombre de 50 años de edad con carcinoma pulmonar broncógeno comprobado, tuvo un considerable derrame pleural he-

morrágico. El estudio del derrame no demostró células de tumor, sino signos de inflamación. Se ejecutó una neumonectomía y se demostró que el derrame fue causado por neumonitis secundaria. El paciente está bien cinco años después de la operación.

La presencia de derrame pleural en casos de carcinoma broncógeno del pulmón no contraindica la operación a menos de que existan signos indudables de metástasis a la pleura, tales como células de tumor en el derrame o tumor visible en la pleura.

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Nutrition Education in Institutions Caring for Tuberculous Patients

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Introduction

Food has always been considered of basic importance in the treatment of tuberculosis. In spite of this, apparently very little systematic effort has been put forth to make the patient conscious of this fact except in those instances when extreme or faddish practices have been recommended.

Nutrition education appears to have been left to chance while the patient was being treated for tuberculosis, according to information gained through individual conferences with clinic and hospital patients and through home visits with nurses during the past five years. A frequent comment made by these patients was "The doctor told me I could go home and lead a normal life." Psychologically this was good advice, but how many of the doctors knew what was "normal life" for his patients, especially when it came to eating? Too often professional people assume that the general population has more information on this topic than it really has. Furthermore practice does not necessarily coincide with knowledge.

A study of the dietaries of 100 patients interviewed in three of the Institute Chest Clinics in Cook County in 1947 revealed that a small percentage of them included the protective foods regularly in their meals. Not one of the arrested cases or their families reported that he had been given any specific instruction regarding his eating habits during the period of treatment. A few had "picked up" and asked for information because they observed that certain foods were served to them regularly while they were in the institutions. One woman had become interested enough in her own food intake to attend nutrition classes given in her community.

Opportunities for Nutrition Instruction

To give the patient an understanding of what food does for him both in sickness and in health, the following methods are available:

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(1) The individual conference is probably the most effective. It is then the patient realizes that someone has a personal interest in him and can help with his particular problems. This is essential to his emotional as well as his physical well being. This method, however, is time consuming, and with the limited staff in most tuberculosis hospitals may not be practical.

(2) The second way to reach a number of patients for educational purposes is to meet with them in small groups, offering all a chance for discussion. The groups need not be large but should be composed of a sufficiently large number to keep the conversation interesting and in useful channels. This method also presupposes that the patients are ambulatory.

(3) The third method for educating patients is the use of literature and visual aids. These must be carefully chosen. Literature should be colorful and easy to read. It must have facts stated simply yet interestingly. Posters should be meaningful and attractive and movies must be timely as well as informational. Though these materials are valuable in teaching patients, their greater contribution is probably that of supplementing and reinforcing the spoken word.

Nutrition Education Programs in Practice

Just how much is done in tuberculosis hospitals to help the patient to have an appreciation of the foods served to him? Because certain impressions regarding nutrition education programs in institutions caring for tuberculous patients had been formed through conferences with the aforementioned patients and their families, it seemed that specific information should also be secured from the other side, namely the sanatorium. A plan was made therefore to study the activities involving nutrition in some of the tuberculosis hospitals and sanatoria in the United States. A brief description of this study is presented.

A two page questionnaire was prepared in cooperation with the statistical section of our organization which when filled out, would give the greatest amount of information, yet demand the least time and effort on the part of the staff members of the institutions who would have to fill them out.

Since this organization is located in the middle western group of states, questionnaires were sent to *all* the tuberculosis hospitals in Minnesota, Wisconsin, Iowa, Illinois, Indiana, Michigan and Ohio. In order to have a larger number and to secure some data for comparison of practices in other parts of the country, copies were also sent to *all* such institutions in Pennsylvania, New York, Colorado and California, and to those under Federal and State control in the remaining states.

Though there are numerous occasions during the course of hospitalization when one might learn of the patient's eating habits and discuss food and nutrition with him, the most obvious times have been recorded and evaluated. These are (1) Upon admittance, when the history and general information are secured. (2) During hospitalization, while collecting and studying dietary records. (3) In making ward rounds when general conferences may be had with the patient. (4) In organized groups for instructional purpose. (5) When distributing literature and presenting visual material.

Another part of this study was to try to determine the kind of literature and posters that had been found most effective in teaching patients. These materials were classified as (1) commercial, (2) those prepared by an organization staff for its own use and (3) those obtained from other noncommercial sources such as the United States DeDpartment of Agriculture, Bureau of Human Nutrition and Home Economics, Local and State Nutrition Associations, State Health Departments and so forth. Because information given on many of the questionnaires was not specific, evaluation of these data was not possible.

Interpretation of Data Returns on Questionnaire

The results from the mailing list described above were gratifying and probably indicated the growing interest in the subject. As shown in Table I, from a total of 452 forms mailed, replies were received from 251. This represents returns of 55.53 per cent. Six letters were returned unclaimed and 195 hospital administrators made no comment or did they return the questionnaire. Thirty-seven letters were received with miscellaneous statements such as explaining that the institution was closed or that they did not

TABLE I

Report of Questionnaire on Nutrition Education and Food Service in Tuberculosis Hospitals and Sanatoria in the U. S. as of August, 1947

RETURNS	*Number*	*Per cent*
TOTAL QUESTIONNAIRES SENT	452	100.0
TOTAL REPLIES:	251	55.53
Questionnaires with adequate information	213	47.12
Questionnaires with inadequate information	1	0.22
Miscellaneous replies	37	8.19
UNCLAIMED MAIL	6	1.33
NO RESPONSE	195	43.14

now take tuberculosis patients. Of the 214 questionnaires returned one had to be eliminated because it could not be classified for lack of information. In the analysis of these data the facts presented are those submitted by 213 tuberculosis hospitals.

Practices as They were Reported

Table II presents a summary of the 213 returned questionnaires. Here we note that a small number of hospital staffs utilized the above mentioned opportunities for nutrition instruction, only 27.2 per cent took dietary histories upon admittance, 39.4 per cent kept dietary records during the period of hospitalization and 39.4 per cent provided special nutrition instruction to their patient population. Of these sanatoria, a number concerned themselves only with those individuals who presented special dietary problems.

Of the institutions that did try to carry on some type of program to instruct the patients those under Federal control were in the majority when checked on a percentage basis. According to Table III, 75 per cent of this group reported that nutrition information was given either through individual conferences, group meetings, distribution of literature or combinations of these. The County and City controlled hospitals were next with 40 per cent participating in such activities. Among the 34 private hospitals questioned only 26.5 per cent indicated they had nutrition education programs.

Among the methods used in teaching patients, individual conferences predominated. Forty-six per cent of the hospitals under Federal control reported that they used this method while 10.7 per cent of them had group meetings, 14.3 per cent combined the group and individual conferences, and 3.6 per cent only distributed nutrition literature. In institutions under other types of control less than 15 per cent reported that they gave nutrition information through individual conferences, while 13.3 per cent of County and City and 7.5 per cent of State hospitals provided group discussions. A small number distributed various kinds of leaflets on the subject. More than 60 per cent of the hospitals, with the exception of the United States government owned, gave no report which probably indicated that there was no attempt made to provide this type of instruction.

Table II also reveals the extent to which the various kinds of materials were used. Considering the combined reports of all hospitals it is obvious that in group three, literature prepared and distributed by the United States Department of Agriculture, State Health Departments and Nutrition Associations was used more frequently than the other two. Again the institutions under Federal control differed from the others in that they used material pre-

pared by their own staff more often than that distributed by either commercial concerns or other official agencies.

Among the users of visual aids, the Federal supported hospitals ranked the highest with 28.6 per cent of them utilizing the educational value of posters and charts and 21.4 per cent showing movies and slides on topics related to nutrition. Only 13.2 per cent of State owned and 10.2 per cent of County and City owned institu-

TABLE II

Summary of Information on Nutrition Education Obtained from 213 Tuberculosis Hospitals and Sanatoria in the U. S. as of August, 1947

TYPE OF CONTROL	DIETARY HISTORY UPON ADMITTANCE		DIETARY RECORDS DURING HOSPITALIZATION		NUTRITION EDUCATION (PATIENTS)	
	Number	*Per cent*	*Number*	*Per cent*	*Number*	*Per cent*
Federal Control:						
Total	28	100.0	28	100.0	28	100.0
For all patients	3	10.7	4	14.3	15	53.6
For specials only	3	10.7	10	35.7	5	17.9
Not reported	22	78.6	14	50.0	8	28.5
State Control:						
Total	53	100.0	53	100.0	53	100.0
For all patients	9	17.0	5	9.4	14	26.4
For specials only	3	5.7	16	30.2	2	3.8
Not reported	41	77.3	32	60.4	37	69.8
County and City Control:						
Total	98	100.0	98	100.0	98	100.0
For all patients	17	17.4	10	10.2	30	30.6
For specials only	6	6.1	25	25.5	8	8.2
Not reported	75	76.5	63	64.3	60	61.2
Private Control:						
Total	34	100.0	34	100.0	34	100.0
For all patients	15	44.1	6	17.7	8	23.5
For specials only	2	5.9	8	23.5	2	5.9
Not reported	17	50.0	20	58.8	24	70.6
For All Types of Control:						
Total	213	100.0	213	100.0	213	100.0
For all patients	44	20.6	25	11.7	67	31.4
For specials only	14	6.6	59	27.7	17	8.0
Not reported	155	72.8	129	60.6	129	60.6

TABLE III

Summary of Methods and Materials Used in Nutrition Education Programs of 213 Tuberculosis Hospitals and Sanatoria in the United States as of August, 1947

	TOTAL		FEDERAL		STATE		COUNTY & CITY		PRIVATE	
	Number	*Per cent*	*Number*	*Per cent*	*Number*	*Per cent*	*Number*	*Per cent*	*Number*	*Per cent*
How Presented:										
Total	213	100.0	28	100.0	53	100.0	98	100.0	34	100.0
Individual conferences	38	17.8	13	46.4	6	11.3	14	14.3	5	14.7
Group meetings	20	9.4	3	10.7	4	7.5	13	13.3	0	0
Combination of above two	15	7.1	4	14.3	3	5.7	5	5.1	3	8.8
Distribute literature only	12	5.6	1	3.6	3	5.7	7	7.1	1	3.0
No report	128	60.1	7	25.0	37	69.8	59	60.2	25	73.5
Literature Used:										
Total	213	100.0	28	100.0	53	100.0	98	100.0	34	100.0
Commercial	11	5.2	4	14.3	1	1.9	6	6.1	0	0
Own	8	3.7	5	17.9	1	1.9	1	1.0	1	3.0
Others*	23	10.8	3	10.7	7	13.2	10	10.2	3	8.8
No report	171	80.3	16	57.1	44	83.0	81	82.7	30	88.2
Visual Aids Used:										
Total	213	100.0	28	100.0	53	100.0	98	100.0	34	100.0
Slides and movies	11	5.2	6	21.4	2	3.8	3	3.1	0	0
Posters and charts	27	12.7	8	28.6	7	13.2	10	10.2	2	5.9
Food models	9	4.2	2	7.1	3	5.7	4	4.1	0	0
No report	166	77.9	12	42.9	41	77.3	81	82.6	32	94.1

*USDA, BHNHE, State Health Departments, etc.

tions made use of posters and charts and approximately 3 per cent of them provided nutrition information by means of movies and slides. A small number of sanatoria used food models of some kind but these were primarily for teaching special diets. Of the 213 tuberculosis hospitals returning questionnaires 171 or 80.3 per cent made no report of literature used and 166 or 77.9 per cent did not comment on the type of visual aids used.

Comments

The information on nutrition education practices obtained through questionnaires sent to hospitals that care for tuberculous patients appears to substantiate the impression gained through interviews with patients as stated in the introduction. Few institutions of this kind have made specific plans to assist the patient to appreciate the part that food plays in treating the disease or in maintaining a healthy body. Of the various types of hospitals studied, those under Federal control apparently have done more towards utilizing opportunities and setting up nutrition education programs than have the others. There has been no attempt made to determine the reason for the practices reported other than to assume that hospitals under Federal control have larger staffs to carry on the program.

A gratifying part of this study was the observation that such a large number of administrators recognized the importance of these activities. Some requested materials and suggestions for initiating nutrition education programs while others said they were planning to start such programs within the next year. There were also many requests for tabulated results of the study.

SUMMARY

A two page questionnaire was sent to 452 tuberculosis hospitals and sanatoria in the United States in order to secure information as to the prevalence of nutrition education programs for patients.

Returns were received from 55.53 per cent of the hospitals of which there were 213 completely filled questionnaires which were used in evaluating nutrition activities.

Of these 213 hospitals approximately one third utilized available opportunities for presenting nutrition information to patients, with only 27.2 per cent taking dietary histories upon admittance, 39.4 per cent keeping dietary records during hospitalization and 39.4 per cent providing organized nutrition instruction.

The individual conference method of teaching was reported as being the most commonly employed by all hospitals. Visual aids appeared not to be in popular use.

Nutrition materials prepared and distributed by such agencies

as the United States Department of Rgriculture, Bureau of Human Nutrition and Home Economics, State Health Departments and State and Local Nutrition Associations appeared to have preference over all others.

Interest in nutrition education for tuberculous patients was evidenced by comments made by administrators and the numerous requests for assistance in initiating such programs and securing materials. Many also were desirous of seeing the results obtained from the questionnaires.

Much of the information obtained in the study has not been incorporated in this article but will probably be presented in other papers at later dates.

CONCLUSION

Nutrition education for tuberculosis patients in hospitals located in all parts of the United States has apparently been neglected according to a study of practices reported by 213 tuberculosis hospitals and sanatoria. Increasing interest in this phase of the program for the care of tuberculous patients is evident. Those employed in the nutrition field might do well to become acquainted with the needs and activities in their particular communities and offer assistance to such institutions.

Acknowledgment: The authors acknowledge with gratitude the cooperation of the hospital administrators in filling out the questionnaires and furnishing valuable information for the study and the assistance given by fellow staff members who so ably prepared and handled the questionnaires, tabulated the data and criticized the paper.

RESUMEN

Se envió un cuestionario que consistía de dos páginas a 452 hospitales y sanatorios para tuberculosos en los Estados Unidos, a fin de obtener datos acerca de la existencia de programas educativos para los pacientes sobre el asunto de la alimentación.

Se recibieron respuestas del 55.53 por ciento de los hospitales, entre las cuales hubo 213 cuestionarios llenados por completo, y estos son los que se emplearon en avaluar las actividades de alimentación.

De estos 213 hospitales la tercera parte, aproximadamente, utilizaron las oportunidades disponibles para presentar a los pacientes información sobre la nutrición, pero sólo el 27.2 por ciento tomaron historias sobre la dieta al tiempo de la admisión, el 39.4 por ciento llevaron datos sobre la dieta durante la hospitalización y el 39.4 por ciento porporcionaron una instrucción organizada sobre la alimentación.

La conferencia individual fue el método de enseñanza que más

generalmente usaron los hospitales. Aparentemente no fue común el empleo de las ayudas visuales.

Los folletos sobre la alimentación preparados y distribuídos por agencias tales como la Oficina de la Nutrición Humana y la Economía Doméstica del Departamento de Agricultura de los Estados Unidos y las Asociaciones de Nutrición Locales y de los Estados, parecieron ser preferidos a todos los otros.

El interés en la educación de los tuberculosos sobre la nutrición fue manifestado por los comentarios que hicieron muchos de los administradores y por las numerosas solicitudes de ayuda para iniciar tales programas y para obtener materiales. Muchos también manifestaron deseos de saber los resultados obtenidos por los cuestionarios.

No se ha incluído en este informe mucha de la información obtenida en el estudio, pero probablemente se la presentará más tarde en otros trabajos.

CONCLUSION

Aparentemente se ha descuidado la educación de los tuberculosos sobre la alimentación en hospitales situados en todas partes de los Estados Unidos, a juzgar por un estudio en el que participaron 213 sanatorios y hospitales para tuberculosos. Es evidente que existe un interés creciente en esta fase del programa de tratamiento de los tuberculosos. Aquellos que están dedicados al campo de la nutrición deberían enterarse de las necesidades y las actividades de sus respectivas colectividades y deberían ofrecer su ayuda a esas instituciones.

Sterile Hemopneumothorax Caused by Softening and Perforation of a Pulmonary Infarct

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Softening of a pulmonary infarct with perforation into the pleural cavity and formation of a sterile hemopneumothorax is unusual. A case demonstrating these events is given in this report. The usual course following pulmonary infarction is organization of the infarct and healing by fibrous scar formation. In some cases a hemorrhagic pleural effusion may develop.

The clinical diagnosis of hemopneumothorax resulting from the breakdown of a bland pulmonary infarct may be exceedingly difficult, especially in the absence of subsequent anatomical studies. No case of this type was encountered in ten years at the Mt. Sinai Hospital of New York City by Berliner,[1] who in 1941 reviewed one hundred and twenty cases of hemorrhagic pleural effusion, ten of which were due to pulmonary embolization. Even the finding of a spontaneous hemopneumothorax is an infrequent finding. Berliner[1] encountered only six cases in a ten year period. Of these, one was due to pulmonary tuberculosis, one was proved to be due to a ruptured bleb, and the others were presumed to have the same cause. Helwig and Schmidt[2] in 1947 reported fourteen fatal cases of spontaneous hemopneumothorax which came to autopsy, but these were all due to ruptured blebs, or torn adhesions, or were of undetermined cause. Similar etiologies have been described in other publications.[5-9].

Rawson and Cocke[3] reviewed the literature in 1947, and found only five cases of perforation of a necrotic pulmonary infarct resulting in hemopneumothorax; they added a sixth case of their own. These cases were diagnosed at necropsy. We regard it worthwhile to report this, the seventh case, because the diagnosis was made during life, conservative treatment proved unavailing, and the clinical impression was confirmed by the necropsy findings.

CASE REPORT

A thirty year old, white male truck driver was admitted to the Salt Lake Veterans Administration Hospital for the third time on December 11, 1947, complaining of severe shortness of breath of two weeks' dura-

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tion. He had been well until four years before entry, when he noted dyspnea, palpitation on exertion, intermittent cough, occasional hemoptyses and orthopnea. A heart murmur was discovered two years prior to entry, although there was no history of rheumatic fever. His first admission to this hospital, thirteen months before the present one, was necessitated by the increasing severity of his symptoms; he was found to have typical evidence of rheumatic heart disease, with mitral stenosis and insufficiency, cardiac enlargement, normal sinus rhythm, and mild decompensation. Rapid improvement followed digitalization. He returned for the second time two months prior to his final admission with a recurrence of his symptoms plus ankle edema. The findings were similar to those found on his first admission. The cardiac rhythm was regular, there was no fever, and repeated blood cultures were negative. Improvement after medical treatment again allowed him to return to work.

Two weeks before the final entry, a sudden transient attack of dysarthria and paralysis of the left extremities occurred. He noticed no fever, petechiae, nor irregularity of the heart. Five days prior to admission a dull aching pain appeared in the right chest posteriorly, radiating around to the front, which was made worse with coughing. The sputum was tinged with blood. Nausea, vomiting, diarrhea and indigestion also occurred, followed by increasing dyspnea, swelling of the ankles, and a yellowish discoloration of the skin.

Physical examination showed an acutely ill, slightly jaundiced, dyspneic and orthopneic young white male. T. 100 degrees F., P. 96, R. 30, and B.P. 125/80. There was an increase in the A-P diameter of the chest with a slight bulging of the precordial region. A respiratory lag was observed on the right. Tactile fremitus was diminished over the right lung. On percussion there was flatness over the right chest posteriorly, below the level of T-10, with hyperresonance above. Auscultation showed

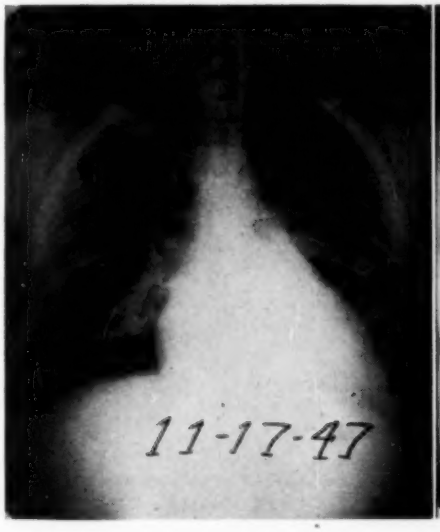

FIGURE 1

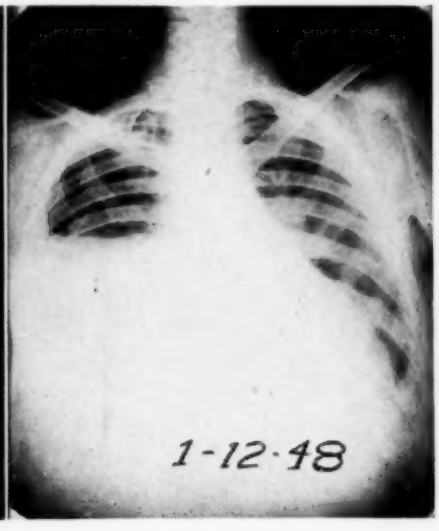

FIGURE 2

Fig. 1: Chest plate taken during patient's second admission. It reveals a mitral configuration of the heart.—*Fig. 2:* Chest film taken on the last admission. Right sided pneumothorax with fluid to the level of the third rib anteriorly. There is also obliteration of the left costophrenic angle.

diminished breath sounds on the right, and coarse rales on the left. A presystolic thrill was felt at the cardiac apex. The area of cardiac dullness was enlarged, the point of maximal impulse was just outside the midclavicular line in the sixth interspace. At the apex a loud, blowing systolic and a low pitched presystolic murmur were heard. The rhythm was regular. P2 was equal to A2. The liver edge was felt four finger breadths below the right costal margin, and there was three plus pitting edema over the lower legs and ankles.

Laboratory work: The urine on admission contained 1 plus albumin and bile while the sediment was normal. Hgb. 17 Gm.; Ht. 51; WBC 18,500 with 80 per cent polymorphonuclear neutrophilic leucocytes, 20 per cent lymphocytes; blood Kahn negative; BUN 11 mg. per cent; prothrombin content 61 per cent; thymol turbidity 4 units; direct bilirubin 0.5 mg. per cent; indirect bilirubin 1.7 mg. per cent. On admission the two hour urine urobilinogen was 4.4 units, rose to 10.8 units three days later, and returned to normal only after several weeks.

Roentgenograms: Films taken on admission (Fig. 2) showed the right lung to be partially collapsed, and there was a large amount of fluid and air in the right pleural space, with shift of the mediastinum to the left. The left costophrenic angle was obliterated. The heart appeared enlarged. Repeat roentgenogram of the chest on the third hospital day following thoracocentesis showed a decrease in the amount of fluid in the pleural space (Fig. 3). A definite disease process in the right lower lobe could be distinguished on the film on the seventeenth hospital day after catheter drainage (Fig. 4).

Repeated electrocardiograms were all normal except for large P waves in Leads I and II. At no time were changes discovered consistent with acute cor pulmonale despite the use of unipolar limb and chest leads in addition to the standard leads.

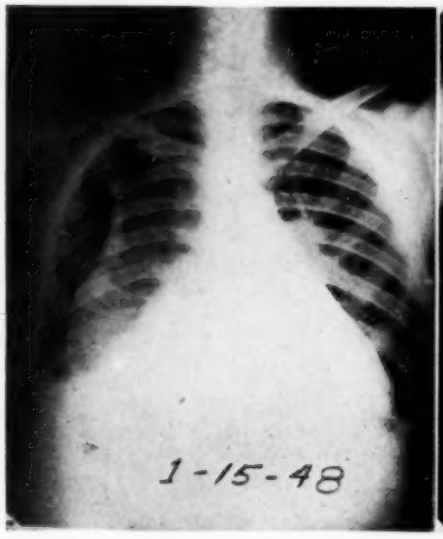

FIGURE 3

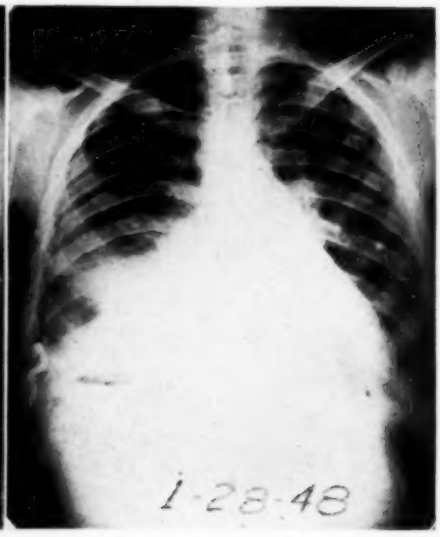

FIGURE 4

Fig. 3: Chest x-ray film subsequent to the removal of 1400 cc. of bloody fluid from the right pleural space.—*Fig. 4:* The presence of right lower lobe pathology is revealed following drainage of the right chest by an indwelling rubber catheter connected to a water trap.

The course in the hospital was as follows: Full digitalization was accomplished. Right thoracocentesis shortly after admission yielded 1400 cc. of red amber fluid with a specific gravity of 1.010. Microscopic examination revealed 200,000 red blood cells per cubic mm., 5,000 white blood cells, mostly polymorphonuclear neutrophilic leucocytes. Culture was negative. No neoplastic cells were seen in a centrifuged specimen. Thoracocentesis was performed again on the fourth hospital day and 600 cc. of red fluid was removed. This had a specific gravity of 1.012. It was loaded with red blood cells and some white blood cells, the majority of which were polymorphonuclear neutrophilic leukocytes. Culture was again negative. Penicillin in water (200,000 units) was introduced into the pleural cavity. By intramuscular route, 500,000 units of penicillin were given every eight hours. The prothrombin content was maintained between 20 and 50 per cent of normal by means of dicoumarol. On the eleventh hospital day, a soft rubber catheter was inserted into the right chest, connected to a water trap and reddish fluid and air under pressure were evacuated. One hundred thousand units of penicillin were instilled into the catheter every other day. Ten days after its insertion, the catheter was clamped off, but the patient promptly developed respiratory distress. A chest film showed further collapse of the right lung (Fig. 5). The catheter was then unclamped and a large amount of air poured out. The patient was placed on a salt-free diet. In addition he received ammonium chloride, mercuhydrin intramuscularly and intravenously, and calcium theobromine salicylate by mouth, all without marked effect on his edema. On January 31, 1948 (25th hospital day), he had an episode of confusion with auditory and visual hallucinations. The patient was placed on six grams of acetysalicylic acid daily without improvement. He continued to have severe hallucinatory episodes and became increasingly dyspneic and cyanotic. His pulse rate was never slower than 90, although intake of digitoxin was pushed to 0.4 mg. daily. On February 21, 1948 all medication except penicillin and 0.1 mg. digitoxin was discontinued. On February 22, 1948 the patient was placed in an oxygen tent because he became too psychotic to utilize an oxygen mask properly. His pulse remained at 100 and was regular in rhythm. His respirations became slow and labored. He became very cyanotic and semi-comatose and quietly expired on the 47th hospital day.

At necropsy (Courtesy Dr. Frank R. Ellis), which was limited to the thorax, the right lung was atelectatic, and the right pleural cavity was filled with air and 50 cc. of brown fluid. Examination of the right lung showed emboli occluding the right superior pulmonary artery, secondary branches of the middle pulmonary artery, and a large branch of the inferior pulmonary artery. In the lower lobe, there was a large infarct measuring 3 x 4.5 x 5 cm., in the center of which was a perforation 1.5 cm. in diameter extending 1 cm. into the parenchyma and communicating freely with the pleural space. An infarct was also present in the upper lobe. The left lung also contained large infarcts peripheral to emboli in the main branches of the arteries to the left upper and lower lobes (Fig. 6). Microscopic examination revealed multiple hemorrhagic infarcts, some showing liquefaction necrosis with large foci of neutrophilic infiltration. One infarcted area revealed many large fusospirochetal rods. These organisms did not grow on cultures obtained at postmortem. There was obliteration of the left pleural cavity by fibrous adhesions, except for two loculated spaces containing brown fluid at the left base.

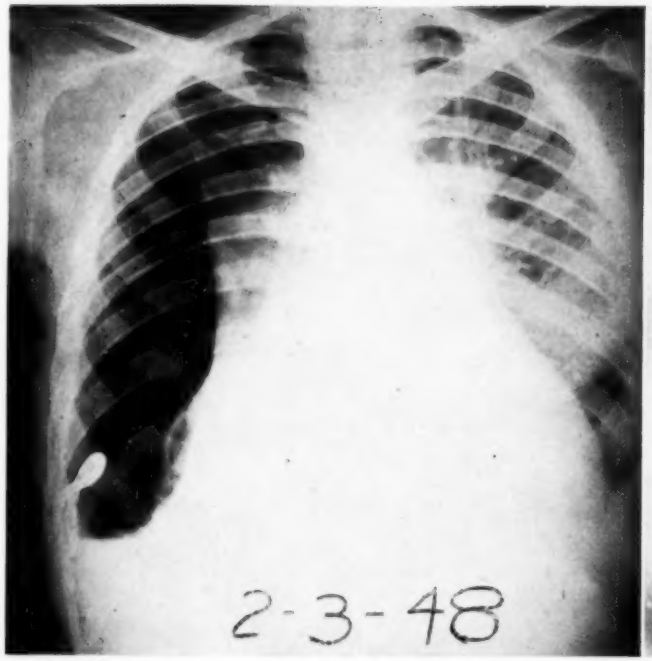

FIGURE 5

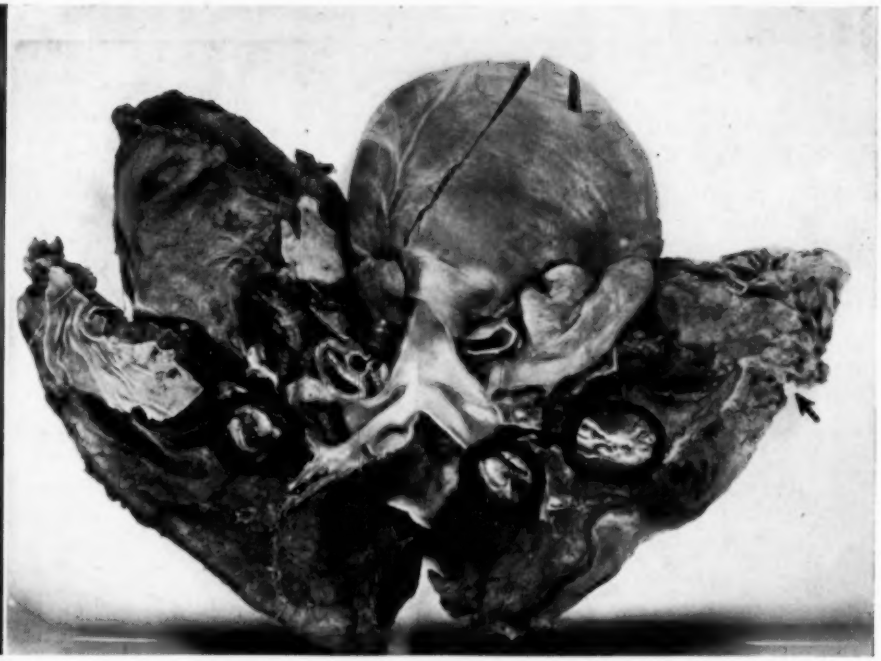

FIGURE 6

Fig. 5: This roentgenogram reveals the rapid development of a tension pneumothorax within several hours after the catheter was clamped off.—*Fig. 6:* The pathologic specimen shows multiple pulmonary emboli with subsequent infarction. The arrow indicates the site of the ruptured infarct. The circles show sites of emboli.

Examination of the heart revealed cardiac enlargement, mitral stenosis with calcified nodular valvulitis, and a small mural thrombus in the right ventricle. There was extensive fibrosis of the myocardium of the right ventricular wall and moderate atrophy and fibrosis of the left ventricular wall. Evidence of active rheumatic changes were not present.

Comments

The antemortem diagnosis of a ruptured pulmonary infarct was made possible by the unusual combination of jaundice and hemopneumothorax. Jaundice has not been reported as a sequel of hemopneumothorax due to the usual causes while icterus as the result of infarction is not at all uncommon. Failure of the pleuropulmonary communication to heal during a period of forty-seven days despite closed drainage mitigated against simple rupture of a bleb or dissection by mediastinal air and favored the presence of a necrotic non-healing area between lung and pleura. In addition, the patient's cardiac decompensation and inactivity certainly predisposed to the formation of thrombi with embolization. The sudden onset of chest pain probably followed an episode of embolization.

Waring[4] includes hemorrhagic pleural effusion under the heading of hemothorax. In this case, pure blood may have been present in the pleural cavity initially, only to be diluted with serous fluid caused by irritation,[1] or effusion due to the decompensation may already have been present when the bleeding took place. Thus, we believe there is justification in the use of the term hemopneumothorax in this case. The fluid was sterile on admission, as shown by the negative cultures, but may have become infected during the long continued catheter drainage. The complete ineffectiveness of apparently adequate dicoumarol therapy in preventing multiple embolization is of interest, as are the failure of the perforation in the right lung to close and the poor response to intensive treatment of the cardiac decompensation. The source of the emboli remains open to question; they may have come from mural thrombi in the heart, but more likely originated in the veins of the legs.

SUMMARY

A case of chronic rheumatic heart disease with decompensation is reported, in which embolization and pulmonary infarction were followed by softening of an infarct, perforation into the pleural cavity, and sterile hemopneumothorax.

RESUMEN

Se informa sobre un caso de enfermedad reumática crónica del corazón con insuficiencia, en el que embolias e infartos pulmo-

nares resultaron en reblandecimiento de un infarto, perforación en el espacio pleural y hemoneumotórax estéril.

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Spontaneous Perforation of the Normal Esophagus

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Spontaneous perforation of the esophagus is a distinct clinical entity, in which, sudden rupture through all layers occurs in the lower end of a normal esophagus. Such a perforation of the esophagus occurs usually after violent retching or vomiting. Acid gastric contents and food particles are driven into the mediastinum, left pleural cavity and also occasionally the right pleural cavity. The resulting insult in this unique esophageal lesion is devastating to the patient. The features of the disease are distinctive yet easily missed thus the diagnosis has been rarely made during life so that definitive treatment could be instituted.

Perforations of the esophagus complicating endoscopy or due to carcinoma, stricture or other pre-existing pathological conditions are not considered in this discussion. The diagnosis of perforation of the esophagus after endoscopy or dilatation, for example, is relatively easy. Similiarly, if the esophagus is the site of a known pathological process, there is little or no diagnostic problem, and treatment may be instituted at once. Perforations due to these conditions are usually less fulminating in character than the lesion under discussion. An empyema or mediastinitis may develop over a period of time and with adequate drainage, chemotherapy and supportive treatment, the patient has a good chance of recovery.

There are several disease syndromes recorded in the literature which are closely related to spontaneous perforation of the esophagus. The lesions in these syndromes are produced by the same fundamental forceful and/or disarranged vomiting mechanism. However, the patient's subsequent clinical course varies and is conditioned by the degree and extent of the resultant esophageal laceration and the possibility of its superimposition on an esophagus already the site of disease.

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A) *Hyperemesis Gravidarum.*

The severe retching and vomiting characteristic of this disease syndrome is well known. Acute longitudinal tears in the mucosa at the lower end of the esophagus may result in severe or fatal hematemesis.

B) *Mallory Weiss Syndrome.*

This syndrome presents fatal hematemesis in an alcoholic following a prolonged debauch with vomiting and retching. The lesion is a longitudinal tear in mucosa or muscularis, usually at the cardia of the stomach and the lower end of the esophagus. Such a laceration in a cirrhotic patient with portal hypertension and esophageal varices may understandably be rapidly fatal from acute blood loss.

C) *True Spontaneous Esophageal Perforation.*

The esophagus is lacerated through all layers, in a longitudinal direction, by vomiting. Gastric juice and food particles are propelled into the mediastinum with consequent gross contamination of it and frequently one or both pleural cavities. In addition air may collect in the mediastinum or pleural cavities under tension and hasten the fatal outcome. This disease entity is the most inexorably ruthless of all esophageal perforations and understandably has carried a one hundred per cent fatal prognosis until very recent times.

We wish to review briefly the clinical aspects of these patients, stress the diagnostic features, mention our laboratory experiments, report a case correctly diagnosed but unsuccessfully treated by surgical means and offer our considered views on the proper surgical and supportive treatment necessary in an attempt to save these patients.

Historical Aspects

In 1724 Boerhaave's classical description of the first case of spontaneous perforation of the esophagus, occurring in the Grand Admiral of Holland, Baron DeWassenaer, was recorded. This has been quoted by Barrett.[1] Fitz[2] in 1877 made the first review in the American literature and recorded a case. In 1914, in a review, Walker[3] found 22 cases including his own, which was the first case diagnosed during life. Other comprehensive reviews include Smead,[4] 1931; Ridgeway and Duncan[5] in 1937; and Klein and Grossman[6] in 1943, who uncovered only 40 cases. Recent reports are those of Collis,[7] 1944; Barrett,[1] 1946; and Eliason and Welty,[8] 1946.

In one of the cases reported by Eliason and Welty[8] the diagnosis was made during life. This represents the fifth time to our knowledge that the correct diagnosis has been made antemortem in this condition. Unfortunately no definitive treatment could be given this patient. Barrett[1] reports three cases in one of which the correct diagnosis was made, but no surgical treatment could be instituted. Collis'[7] patient was a healthy 45-year-old soldier on whom a thoracotomy was performed, the esophageal rent closed and the pleural cavity drained, but despite this definitive treatment, the patient succumbed ten hours later.

Recently Barrett[9] has reported the first case known to us of survival of a spontaneous perforation of the esophagus. His patient had a subtotal hysterectomy then vomited violently 13 days postoperatively. Diagnosis was made by thoracentesis and roentgen examination and operation performed ten hours later. The esophageal perforation was closed by a purse-string suture and the chest drained. The immediate course was good. Three weeks later, a mediastinal abscess required drainage. Two months later, an abscess of the right upper lobe was noted, however, this drained spontaneously and the patient was well thereafter. Our colleague, Doctor Joseph P. Lynch,[10] has likewise successfully treated a classical case recently by early operation and suture of the esophageal perforation, as outlined in this study. This case will be reported in detail at a later date by him.

Etiology and Pathogenesis

In spontaneous perforation, the stage is usually well set by a fairly standard set of circumstances. The patient usually has a full stomach at the time he retches violently, gastric contents are ejected exerting great expansile pressure on the upper gastric and lower esophageal wall, with resultant rupture of the distal esophagus. If the esophagus is obstructed even transiently by a bolus of undigested food leading the column of vomitus, the possible mechanism becomes even more apparent.

Rupture occurs in the lower end of the esophagus apparently due to an inherent weakness of this anatomic area. MacKenzie, quoted by Smead,[4] removed the esophagus from 18 specimens at postmortem examination and ruptured them by water pressure. In 17 cases, the tear occurred in the lower third of the esophagus with from 5 to 11 pounds pressure.

A congenital and anatomic weakness of the lower end of the esophagus has been postulated by Menne and Moore[11] on the basis of their case of spontaneous esophageal perforation in a five-month-old infant following repeated vomiting spells. A curd stuck in the infant's esophagus and throat. The bady vomited

once more following this and died of a typical ruptured esophagus thirty-six hours later. They state that there are three other authentic cases in the literature over a 200-year period occurring in infants and children.

There is a strong alcoholic history in many of these patients. Ridgeway and Duncan[5] reported 18 of 35 cases as having a significant alcoholic history; 7 cases denied indulgence and 10 cases were questionable or not stated. They felt, therefore, that regurgitation of gastric juice with digestion and/or acute ulceration might very well be a confusing etiological factor and make it difficult to evaluate microscopic changes in the esophagus as to their occurrence before or after rupture.

Alcohol may be an important factor in other ways. Excessive amounts often produce retching and vomiting and also tend to produce incoordinated muscle action that may impair critically the complicated vomiting mechanism.

Gluttony may be a factor since the full stomach is perhaps more easily compressed between the diaphragm and abdominal wall.

According the Weiss[12] vomiting is a complex reflex act composed of a number of highly coordinated bodily changes that he describes as follows:

(1) During the stage of nausea, the pylorus closes and the cardia and esophagus dilate. (2) Reverse peristalsis carries gastric contents to the cardia and esophagus. (3) At the onset of retching, the diaphragm descends to the position of deep inspiration. (4) Gastric contents are then ejected with great force by this sudden convulsive increase of intrathoracic and intra-abdominal pressure. (5) Repeated attacks of vomiting at short intervals, however, may fatigue the vomiting center with consequent loss of coordination. (6) Gastric contents may then thrust with even greater force against the cardia and lower esophagus if relaxation of the esophagus and descent of the diaphragm do not occur at a properly timed juncture with the forceful retching movement.

Pathology

1) Postmortem examination of a patient who has died as a result of spontaneous perforation of the esophagus reveals a remarkably constant and characteristic lesion. The rupture almost invariably occurs in the lower one-fifth of the esophagus, in a longitudinal direction and on the left posterolateral aspect. The tear is usually sharp edged and incised in appearance. It may be several centimeters in length but may be small and difficult to demonstrate. Rarely the rupture has been reported as occurring transversely, on the right, high in the esophagus, or below the diaphragm. The appearance in microscopic sections varies with

the survival time of the patient and the interval between death and postmortem examination. The majority of pathologists seem agreed that rupture occurs in the "true" case in a normal esophagus, that there is no evidence of pre-existing disease, and that all tissue changes are due to the severe chemical and bacterial inflammation.

2) There are, immediately, several possible mechanisms resultant upon a spontaneous rupture of the esophagus: (a) Rupture into the mediastinum and left pleural cavity which is the commonest lesion. (b) Rupture into the mediastinum and both right and left pleural cavities. (c) Rupture into the mediastinum and right pleural cavity only. (d) Rupture into the mediastinum alone or with later perforation into either pleural cavity. (e) Direct rupture into the mediastinum and/or pleural cavity. A devastating, necrotizing chemical insult with rapidly superimposed aerobic and anaerobic infection quickly jeopardizes the patient's life and he succumbs to the gangrenous mediastinitis, pleuritis, pyopneumothorax, and emphysema usually in a matter of several hours to 1 or 2 days. The pyopneumothorax may exhibit signs of tension, varying degrees of atelectasis are present, and mediastinal emphysema may cause thoracic inlet obstruction. The latter impairs inflow to the right heart with consequent peripheral vascular collapse and profound shock. Varying amounts of fluid and gastric juice, often bloody, together with partially digested food particles will be found in the mediastinum and/or pleural cavities. A pericardial effusion may ensue if the patient survives for any length of time.

Symptoms and Signs

The catastrophe of spontaneous esophageal perforation is usually seen in a male between 35 and 55 years who had a history of heavy or excessive alcoholic intake. Finally, one of these indulgences is accompanied by severe retching or vomiting and at this time rupture of the esophagus is particularly apt to occur.

In Walker's[3] series of collected cases, 20 of 22 patients were males. Only 5 of the 35 cases reported by Ridgeway and Duncan[5] were females. According to Menne and Moore[11] 3 authentic cases have been reported in children in addition to their case.

Vinson[13] has reported several cases in which pernicious vomiting of pregnancy caused acute lacerations in the lower end of the esophagus later resulting in a stricture if the patient survived. These cases are undoubtedly counterparts of the cases reported by Mallory-Weiss.[14] In the Mallory-Weiss syndrome, vomiting and retching occurs in chronic alcoholics during a prolonged debauch leading to hematemesis from a long tear through the mucosa or

even into muscularis of the lower esophagus or gastric cardia. Indeed, one of the patients reported by Mallory and Weiss was a hyperemesis gravidarum case with an identical lesion to that observed in alcoholics. The cases of Vinson and Weiss and Mallory may be regarded as incomplete ruptures of the esophagus.

Cases have also been reported as occurring during a convulsive seizure, defecation, seasickness and retching during or following recovery from general anesthesia.

In most instances, the onset of symptoms is sudden, and the patient rapidly becomes acutely and seriously ill. Usualy after vomiting or retching, the patient is suddenly seized with violent pain and may volunteer that he feels as though something was "torn inside."

Pain is continuous and excruciating, and may be felt low in the chest, over the xiphoid, may radiate through to the back, or may be referred to the shoulder or upper abdomen. Morphine or demerol even in huge doses do not alleviate the patient's discomfort.

Shock is invariably present. The patient is anxious and apprehensive. He may be restless or may remain immobile with trunk flexed. There may be slight cyanosis and the respirations are usually rapid, shallow and grunting. The skin is cold and clammy, the blood pressure low, the temperature normal or sub-normal and the pulse fast and thready.

Thirst is insatiable, although pain may be intensified as swallowed fluid passes into the mediastinal and pleural spaces.

Upper abdominal spasm and tenderness may be prominent before the chest findings are manifest. Exploratory laparotomy has been performed frequently in the past for this reason.

The Chest findings include signs of fluid relatively early. Later, atelectasis and pyopneumothorax are present. The pneumothorax may manifest signs of tension by dyspnea, cyanosis and displacement of the mediastinum to the opposite side.

Subcutaneous Emphysema is a very important and helpful sign and should lead one to the diagnosis if present. Usually, it is present in two-thirds of the cases, appearing first in the suprasternal notch and supraclavicular fossae, and later spreading so as to involve the face, trunk or even the extremities and dissecting widely in the subcutaneous tissue.

Thoracentesis will clinch the diagnosis in the suspected case. Varying amounts of air may be aspirable. The fluid present usually is sour and in appearance often seems tinged by old changed blood, presumably due to the action of gastric ferments. A thin serous fluid may be present in either pleural cavity if

the infection is confined to the mediastinum. A few hours later this fluid likewise will be invaded by organisms.

Roentgenography may be extremely helpful and is important in the differential diagnosis and as a possible means of early diagnosis. The roentgenogram may confirm the signs of fluid in one or both pleural cavities, demonstrate atelectasis or pneumothorax and corroborate mediastinal displacement. Emphysema may be detected in the root of the neck or in the mediastinum. If rupture has occurred only into the mediastinum, a fluid level may be demonstrated. The absence of air under the diaphragm is helpful if there is doubt as to the location of the primary lesion.

Many of the classic findings in perforated esophagus may be evident only shortly before the patient succumbs. However, if the diagnosis is suspected, thoracentesis and suitable roentgenograms will often solve a perplexing differential problem long before emphysema appears. The difficulty now is not really in making the diagnosis but rather in making it early enough to have definitive surgery offer some chance to the afflicted individual.

Differential Diagnosis must include: (1) acute coronary thrombosis, (2) perforated peptic ulcer, (3) acute pancreatitis, (4) dissecting aneurysm of the aorta, (5) acute cholecystitis, (6) spontaneous pneumothorax, (7) pulmonary embolism, (8) mesenteric occlusion, (9) interstitial emphysema, (10) diaphragmatic hernia.

Laboratory Experiments

Following the unsuccessful case herein reported the production in the laboratory of a lesion exactly comparable to a spontaneously ruptured esophagus was attempted. The creation of an esophageal defect by various methods through the esophagoscope was unsatisfactory because a truly comparable lesion could not be produced and no two perforations by such methods were alike; the mediastinum was not widely opened nor even necessarily contaminated and the right pleural cavity was not opened. Such procedures were, therefore, abandoned.

Eventually, in order to produce a standard defect analogous to the human case, a thoracotomy was done and the lesion produced under direct vision in spite of the obvious disadvantages of assessing therapy on an animal who has had a previous thoracotomy. A standard procedure was used in each animal. The animal was fed then anesthetized with intravenous nembutal, shaved and prepped, an endotracheal tube inserted, and the animal placed in the right lateral decubitus position. The chest was opened through a left intercostal incision in the seventh interspace. The esophagus was mobilized up to the arch and the right and left pleural cavities and mediastinum widely opened. A two-inch left

postero-lateral incision through all esophageal layers was then made approximately 1½ inches above the esophageal hiatus. Ten milligrams of apomorphine was given then intravenously supplemented by manual compression of the animal's full stomach. Large amounts of gastric juice and previously eaten food were regurgitated with great force into the mediastinum and both pleural cavities. The lungs were expanded under positive pressure, air was aspirated from the right and left pleural cavities while the chest wall was closed in layers. A drainage tube was led out to an underwater seal. In this fashion, a lesion was produced analogous to the human case in its most severe form, namely, with both pleural cavities, as well as the mediastinum, widely involved by gastric juice and food particles.

The animals were divided into three groups: (a) completely untreated, (b) supportive treatment by parenteral fluids, sulfadiazine and penicillin, (c) operative closure of the esophagus at varying intervals and pleural drainage with parenteral fluids, penicillin and sulfadiazine.

GROUP I — UNTREATED

Animal	Survival	Post Mortem Findings
Dog No. 1	40 hours	Putrid empyema and mediastinitis.
Dog No. 2	1 hour	Mediastinal emphysema. Pneumothorax.
Dog No. 3	18 hours	Putrid empyema and mediastinitis.
Dog No. 4	Less than 1 hour	Mediastinal emphysema. Tension pneumothorax.
Dog No. 5 (unfed)	Survived	
Dog No. 5 (fed)	7 hours	Previous esophageal lesion firm fibrous tissue repair. Many adhesions. Recent esophageal laceration. Empyema and mediastinitis.

These animals were intended to serve mainly as a control group. An attempt was made to restore their cardio-respiratory physiology to normal as the chest was closed, but no further treatment was given. As might be expected, varying degrees of pneumothorax and mediastinal emphysema resulted. Varying amounts of serosanguinous fluid was found in the pleural cavities. This fluid invariably became thicker and more foul the longer the animal survived. In Dog No. 5 (unfed) there was no gross contamination by food or gastric juice. The edges of the esophageal lesion bled

freely and there was a large clot in the left pleural cavity as the chest was closed. Persistent leakage was prevented probably by this fact. In any event, the animal survived and 3 months later, the chest was re-opened. The site of previous laceration was firmly repaired but the scar was apparent on careful palpation. There were numerous adhesions, however, and the examination was otherwise not remarkable. This case presumably illustrates the importance of the gastric juice and resultant chemical pleuritis as a factor of this lesion, particularly in view of the low PH values of the dog's gastric juice. When this animal's esophagus was again lacerated, this time on a full stomach, death occurred in the usual fashion 7 hours later.

GROUP II — SUPPORTIVE TREATMENT

Animal	Survival	Post Mortem Findings
Dog No. 6	18 hours	Putrid empyema, mediastinitis, minimal mediastinal emphysema, partial atelectasis, left lower lobe.
Dog No. 7	18 hours	
Dog No. 8	18 hours	

These animals all received 400-800 cc. of 10 per cent glucose in saline intravenously according to their weight. Two hundred thousand units of penicillin were placed in the chest before closure. Sodium sulfadiazine (1 grain per pound) was mixed in the intravenous fluids at the conclusion of operation. Penicillin was then given in a dose of 100,000 units intramuscularly every eight hours and sodium sulfadiazine (1 grain per pound per 24 hours) was given subcutaneously every eight hours. The intravenous fluids were repeated 12 hours following operation. Postmortem examination revealed the findings charted above.

Group III: Operative and Supportive Treatment

A thoracotomy was performed and the usual esophageal laceration produced. Eight hours after perforation (to allow for diagnosis in the human case) fluids, sulfadiazine and penicillin were begun. Animals Nos. 9, 10, 11, and 12 were then re-operated upon at intervals after perforation of 16, 11, 8 and 8 hours respectively, and the perforation closed. In all these animals, the chest was re-opened by subperiosteally resecting the left ninth rib. The pleural cavities were thoroughly irrigated. The esophageal rent was closed by an inverting Connell suture of 1-0 chromic catgut. The muscular layer was closed by multiple interrupted Lembert sutures of 3-0 silk. A drainage tube was led out through the tenth interspace and connected to an underwater seal. The

chest wall was closed in layers. Supportive therapy was then continued by the same schedule outlined for Group II. These animals died, 24, 15, 10, and 10 hours postoperatively, respectively. In animal No. 12 only the left pleural cavity was opened at the time of perforation of the esophagus with no apparent change in the end result.

Animal No. 13: This animal had his esophagus perforated. Eight hours later supportive treatment was instituted and twenty-four hours later, he was re-operated and the esophageal lesion closed in two layers. The animal did very well until the seventh post-operative day when he became distended and died a few hours later. Postmortem examination revealed an essentially normal chest cavity with the esophagus well closed. Abdominal examination revealed generalized peritonitis.

Then in an attempt to evaluate the time element and the trauma of a previous thoracotomy, the esophagus in three additional animals was perforated. The esophageal laceration was sutured immediately. Two of these animals survived.

Animal No. 14 and animal No. 16: The esophagus was perforated and the mediastinum and pleural cavities contaminated forcefully by vomitus, then thoroughly irrigated with saline which was aspirated. The esophageal rent was closed at once in two layers. Chemotherapy was stopped 24 hours later, and the chest drainage tube removed. The subsequent course of these dogs was completely uneventful.

Animal No. 15: The esophagus was perforated in the usual manner and immediately sutured using the same technique as

GROUP III — SUPPORTIVE AND OPERATIVE TREATMENT
Esophageal Perforation Closed at Varying Intervals

Animal	Closure Esophageal Perforation	Survival	Post Mortem Findings
Dog No. 9	16 hours	40 hours	Esophagus well closed. Pleural and mediastinal inflammation. No suppuration.
Dog No. 10	11 hours	26 hours	
Dog No. 11	8 hours	18 hours	
Dog No. 12	8 hours	18 hours	
Dog No. 13	24 hours	7 days	Peritonitis.
Dog No. 14	Immediate	Survived	
Dog No. 15	Immediate	24 hours	Putrid empyema and mediastinitis.
Dog No. 16	Immediate	Survived	

Dogs 9-13 inclusive had two thoracotomies.

in the other animals. In spite of the usual postoperative measures, the animal died twenty-four hours later. Postmortem examination revealed the esophagus to be well closed but putrid empyema and mediastinitis were present.

Comment

The severest possible form of esophageal perforation was produced with contamination of the widely opened mediastinal and pleural cavities. No absolute conclusions can be drawn from such a small series of dogs, however, the results are suggestive.

The group of animals with no treatment and with supportive care only show no great variation in time interval between perforation and the expected result. The animals, in whom the laceration was sutured at a subsequent operation, tended on the whole, to live a few hours longer and one of these animals, with a 24-hour interval between laceration and suture of the perforation, lived seven days and then did not die of chest complications. Three animals had their esophageal perforations closed immediately. The subsequent course of two of these animals was uneventful. The other animal, however, died of continuing mediastinal and pleural infection. The prolonged or complete survival of an animal with so severe a lesion seems to us directly related to the closure of the rent and the consequent prevention of continued contamination of the mediastinal and pleural cavities.

In view of the known disastrous course in the human patient with an untreated esophageal perforation and the uniformly fatal course in all dogs whose closure was delayed longer than the immediate termination of the acute experiment, it is difficult to explain the survival times of dogs nine to thirteen inclusive since these were in inverse relation to the interval between perforation and cuture. The obvious explanation would seem to be the increased interval between the thoracotomies performed first to create the lesion and then to institute treatment in each animal. These animals surviving longest had the greatest time interval in which to recover from the primary thoracotomy. The animals in whom suture was immediately performed are more strictly comparable to human cases and to Groups I and II inasmuch as only one thoracotomy was performed. The essential difference is closure of the esophageal laceration in these comparable cases, with a change from 100 per cent mortality to 67 per cent survival. In this group of dogs, Nos. 9-16 inclusive, who were treated ideally with time intervals approximating the problem in the human, the survival rate was such (2 out of 8) that we believe that the results of the experiments suggest that the rationale of treatment is justified.

CASE REPORT

This 53-year-old laundry worker entered Boston City Hospital for the first time on May 7, 1946.

Chief Complaint: Excruciating epigastric pain for six hours.

Present Illness: Patient had never been seriously ill prior to this admission. At 6:30 P.M. on May 7, he ate lamb stew. The entire family became nauseated and the patient vomited mildly at 7:00 P.M. He then attended a club meeting where he drank two bottles of beer. He returned home at 9:30 P.M. feeling ill, vomited repeatedly, and shortly experienced:

(a) Severe, sharp, high epigastric and low left chest pain. The pain began anteriorly and radiated around to the back and also to the left shoulder.

(b) Sweating with a cold clammy feeling.

(c) Vomiting of clear gastric juice without blood.

At 1:30 A.M. the patient was seen in the emergency ward and given one-quarter of a grain of morphine sulfate on the admitting floor and sent to the ward with a probable diagnosis of acute coronary thrombosis.

Physical Examination: Temperature, 98 degrees F.; pulse, 140; respiration, 32; blood pressure, 130/90. Inspection revealed a very apprehensive, obese, middle aged male who was cyanotic, clammy, and in acute excruciating pain in spite of morphine. Respirations were rapid and painful. The patient was twisting and turning on the litter and complaining of thirst. His pupils were pinpoint. Chest examination was negative on percussion and auscultation except for high diaphragms. The heart not enlarged. Normal sinus rhythm at a rate of 140. The abdomen was boardlike with high epigastric tenderness. No peristalsis was heard. The reflexes were hyperactive.

Laboratory Data: Hgb., 90 per cent; WBC, 20,800; Polys, 96; Lymphs, 4; Platelets and RBC, normal.

Hospital Course: The patient continued to complain of thirst and of excruciating pain. He received one-half grain of morphine, one grain of codeine and one hundred milligrams of demerol in a period of two hours with no relief. The electrocardiogram was of no significance. The urinary diastase was normal. The flat and sitting abdominal roentgenograms revealed no fluid levels or air beneath the diaphragms. A general surgical consultant believed the abdomen was not a surgical problem. Chest roentgenograms revealed elevation of the left diaphragm and a hazy opacity at the left base. There was no fluid level. (Later review of this film revealed emphysema of the mediastinum). The patient was maintained on nasal oxygen, demerol and penicillin. At 8:00 A.M. on May 8, the patient's blood pressure was 100/70 and the abdomen was rigid. Neck swelling with crepitation was present and air was beginning to dissect in the tissue planes of the face. A diagnosis of mediastinal and subcutaneous emphysema was made. A portable chest x-ray film revealed a partial collapse of the left lung, mediastinal shift to the right with fluid obscuring the left lung field, elevation of the left diaphragm and subcutaneous and mediastinal emphysema. The Thoracic Surgery Service was called in consultation and the patient seen by one of us (J.W.S.) and a diagnosis of spontaneous perforation of the esophagus was made. A thoracentesis revealed fluid with food particles and low PH and the diagnosis was thus confirmed.

The patient's condition at this time was extremely precarious and it

was felt that he would tolerate little if any operative manipulation. A whole-blood transfusion was started, then under local one per cent procaine infiltration, a closed intercostal catheter drainage was performed in the seventh left interspace in the posterior axillary line on May 8 at 12:00 A. M. A large amount of extremely foul thin cloudy fluid together with a large amount of air was immediately aspirated on opening the catheter to the underwater seal bottle. The patient was then transferred to the ward in poor condition. Intravenous fluids and blood were given. A positive pressure mask was used to deliver oxygen. Penicillin was given in doses of 30,000 units intramuscularly every two hours. Sodium sulfadiazine was given intravenously once then discontinued because of the poor urinary output. In spite of medication, the patient was obviously in severe pain; he was perspiring profusely; his color was only fair even with positive pressure oxygen. His pulse was weak and thready with a rate of 116 to 140; blood pressure remained at shock levels. Abdominal distention made it even more difficult for the patient to aerate properly. The patient was catheterized, approximately 20 hours after admission, and fifty cubic centimeters of highly concentrated urine was obtained. His non protein nitrogen was 76; total protein, 6.56; carbon dioxide, 39 per cent; red blood count, 4,000,000; and white blood count, 17,500. A right thoracentesis revealed forty cubic centimeters of cloudy serous fluid. Direct smear and culture of this fluid were negative. Penicillin was instilled, however, the patient's condition remained the same throughout May 8, and early May 9, 1946. He was extremely restless, complaining almost constantly of pain and asking for water. His skin and extremities were mottled and cyanotic and the skin was cold and clammy. The patient was disoriented and irrational and difficult to restrain.

At 9:30 A. M. on May 9, 1946, the patient's temperature suddenly rose to 105.6 degrees F. by rectum where as his previous temperature had been 99 degrees F. At this point, it became extremely difficult to count his pulse and his respirations rose to 40-44 and his blood pressure fluctuated slightly but always at shock levels. He was continued on a supportive therapy. An attempt was made to restore his blood chemistry to normal. The patient was obviously going rapidly down-hill from extensive and overwhelming sepsis.

At 2:00 P. M. on May 9, 1946, the patient was taken to the operating room and under local procaine infiltration, a posterior mediastinotomy was done as a desperate measure in a nearly moribund patient in profound shock. A four-inch paravertebral incision was made. The erector spinae muscle group was retracted. Two-inch segments of the 9th and 10th ribs were removed subperiosteally and the posterior mediastinum was entered. There was evidence of extensive mediastinal infection with a large rent present in the left pleural membrane. There was a small similar tear in the right pleural cavity: both of these openings were widely enlarged for free drainage. The actual point of perforation of the esophagus could not be determined. The cavity was then packed with hydrogen peroxide sponges followed by a dry sterile dressing and adhesive strapping. The closed intercostal drainage tube was kept in place. Immediately after this procedure the patient seemed much relieved; he was, of course, returned to the ward with an extremely poor prognosis.

One hour after his return to the ward his condition deteriorated. Respirations were 40; blood pressure was unobtainable, and his pulse

could not be counted. A continuing effort to elevate the blood pressure was made with whole-blood transfusions; this continued to be unavailing and oxgenation by mask was inadequate. Two hours after his return from the operating room there was no response to stimulants and the patient expired at 4:15 P.M. on May 9, 1946.

Three hours later, post mortem examination revealed a longitudinal laceration of the esophagus in its distal portion on the left posterolateral aspect approximately one-half centimeter above the cardia. B. Coli Communis was cultured post mortem from the heart's blood and right and left lower lobes. There was dissection of the esophagus 15 cm. upward above the laceration and air bubbles in the surrounding mediastinal tissues. Pressure on the stomach pushed gastric contents into the left pleura.

Microscopically, the esophagus, at the point of rupture, revealed an acute inflammatory reaction with tissue necrosis and blood vessel thrombosis extending through even the outermost layers and involving the peri-esophageal tissues as well. The laceration was approximately three centimeters in length and associated with (1) intramural and peri-esophageal abscess formation, (2) severe mediastinitis with mediastinal emphysema, (3) bilateral severe pleuritis, (4) subcutaneous emphysema, (5) passive congestion liver, (6) atelectasis left lower lobe, (7) minimal pericardial effusion.

Treatment

Following a spontaneous perforation of the esophagus, the patient's course generally is progressively and rapidly downhill. Time is of the essence in undertaking to treat this condition. The most important single factor is the time interval between the occurrence of the rupture and the placing of the patient in the hands of the Thoracic Surgeon. It is our belief that operative maneuvers such as closed intercostal drainage and posterior mediastinotomy are measures of procrastination and will probably not be successful in saving one of these patients except under very unusual circumstances. A bona fide case of spontaneous perforation of the esophagus with fulminating sequelae will run a rapidly fatal course in spite of measures such as these. These patients almost from the moment they are first seen, are so seriously ill, so obviously prostrated and so nearly moribund that one's first impulse is to consider their prognosis hopeless and to undertake only to make them comfortable insofar as possible. This policy in the past, aside from the problem of diagnosis, has led to a 100 per cent mortality. Regardless of the condition in which the patient is first seen, we believe that it is of the utmost importance to quickly render the patient whatever supportive treatment is possible and undertake definitive treatment at once. No matter how acutely ill the patient appears at that time, he will show no further improvement and within a matter of hours his condition will rapidly deteriorate as signs of sepsis and toxicity develop.

To our minds then the procedure of choice in a spontaneous perforation of the esophagus is an open thoracotomy with endotracheal anesthesia, adequate oxygenation and positive pressure as indicated. The mediastinum should be widely opened and decompressed into the left pleura. If the right pleura is grossly contaminated this should also be widely opened. The laceration of the esophagus can be rapidly closed with an inverting mucosal suture of catgut followed by interrupted silk sutures, thus obviating further contamination. The anesthetist should then expand the lungs; the right chest should be sucked out during this process and the chest closed tightly with the left pleura drained by an adequate underwater seal drainage system. The cardio-pulmonary physiology is thus restored to as nearly normal a situation as possible and the patient is given his maximum possible chance to combat infection.

Supportive measures as an adjunct to thoracotomy: (1) *Oxygen* delivered either by nasal catheter or preferably by a positive pressure mask. (2) *Adequate fluids parenterally* including the liberal use of whole-blood. (3) *Massive doses,* 200,000 to 500,000 units, of *penicillin* in both the right and left pleural cavities and in addition 100,000 units of penicillin intramuscularly every three hours should be given. (4) *Streptomycin* may be given during the acute phase probably in a dose of 0.5 grams every six hours to help combat the mixed infection realizing that later the organisms may develop resistance to the drug. (5) *Sulfadiazine* therapy may or may not be practical parenterally depending upon the urinary output. (6) The *patient* should be made as *comfortable* as possible by adequate doses of demerol although morphine may be necessary. (7) *Nothing by mouth* for 24 to 36 hours. (8) The *mouth and throat* should be sprayed with penicillin solution, 10,000 units to the cubic centimeter giving approximately 1 cc. every two hours until the esophageal laceration is closed. At the same time, careful mouth care should be given to the patient. (9) *Abdominal distention* may be combated by the usual measures, flaxseed poultices or hot-water bottles, prostigmine, and rectal tubes. (10) If the *left pleural cavity* is drained, the right pleural cavity should be considered as potentially infected even if no tear exists and a *right thoracentesis* should be done once or twice daily as indicated by the portable chest roentgenogram and intrapleural antibiotics given. Irrigation of the drainage catheter through a Y tube may be carried out every two hours if necessary. Penicillin and streptomycin diluted in saline should be used as the irrigating fluid. (11) If *mediastinal emphysema* is severe with paradoxical pulse and obstructed inflow to the right heart, a simple collar incision may be done as the patient lies in bed.

A finger introduced into the anterior superior mediastinum will suffice to effect decompression. (12) *Competent nursing* care is essential.

SUMMARY

1) Spontaneous perforation of the esophagus is a distinct clinical entity, in which, sudden rupture through all layers occurs in the lower end of a normal esophagus.

2) The diagnosis has been rarely made during life so treatment could be instituted. The mortality until very recently has been 100 per cent.

3) Rupture occurs usually in a middle aged, alcoholic male.

4) The laceration classically occurs in the lower one-fifth of the esophagus, in a longitudinal direction and on the left posterolateral aspect and is sharp edged and incised in appearance. Mediastinitis, empyema and pneumothorax usually result immediately.

5) Chest roentgenograms, thoracentesis and subcutaneous emphysema will enable one to make a correct diagnosis.

6) A case report of a patient unsuccessfully treated is presented.

7) On the basis of laboratory experiments a rationale of treatment is suggested involving supporting care with early thoracotomy and suture closure of the laceration.

Note: The authors wish to express their appreciation to Doctor Stephen S. Maddock, Director of the Surgical Research Laboratory of the Boston City Hospital for his cooperation, and advice in this study.

RESUMEN

1) La perforación espontánea del esófago es una entidad clínica clara, en la que la ruptura repentina, a través de todas las capas, ocurre en la extremidad inferior del esófago normal.

2) Sólo con rareza se ha hecho el diagnóstico durante la vida para permitir que se aplicara el tratamiento. Hasta muy recientemente la mortalidad ha sido del 100 por ciento.

3) Generalmente la ruptura ocurre en un hombre alcohólico de edad mediana.

4) Clásicamente, la ruptura ocurre en el quinto inferior del esófago, en una dirección longitudinal y en el aspecto posterolateral izquierdo, y tiene un borde agudo y que parece cortado. Mediastinitis, empiema y neumotórax generalmente resultan inmediatamente.

5) Los roentgenogramas torácicos, la toracentesis y el enfisema subcutáneo facilitan el diagnóstico correcto.

6) Se presenta un informe sobre un caso que fue tratado con mal éxito.

7) A base de experimentos de laboratorio se sugiere un tratamiento que consiste de cuidado sustentante, con toracotomía temprana y sutura de la laceración.

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Chronic Constrictive Tuberculous Pericarditis* Report of a Case with Pericardiectomy

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As its name implies, this disease is distinguished by an inflammatory and cicatricial transformation of the pericardium, trapping the heart in a compressive, inelastic envelope. The deposition of scar tissues may incarcerate the heart, fix the heart and pericardium to unyielding surrounding structures and constrict the great veins and aorta. Clinically, the predominance of any or all of these features will have its peculiar bearing on the disease.[5] The cumulative result is circulatory failure from deficient filling of the heart. The problem seems to stand clear in its simplicity—the removal of a mechanical obstruction, a familiar task to the surgeon. Unfortunately, the location of the involvement, its etiology and the wretched condition of the patient makes this task formidable indeed.

The organism held frequently responsible for constrictive pericarditis is the tubercle bacillus.[13] Blalock and Burwell[2] demonstrated a tuberculous etiology in 18 of 28 cases. Three cases were due to pyogenic organisms. Harrington[6] found tuberculosis in 5 of 21 cases. Rheumatic pericarditis rarely progresses to the chronic constrictive stage and therefore rheumatic fever cannot be incriminated. Tuberculous pericarditis usually occurs in association with tuberculosis of the lymphatics, pleura or peritoneum. The pericardium commonly becomes involved by extension of adjacent infection from caseous mediastinal or peribronchial lymph nodes. The dynamics of constrictive pericarditis follow the familiar pattern of tamponade. They were clearly described by Burwell and Blalock[4] as: (1) High peripheral venous pressure. (2) Low peripheral arterial pressure. (3) Basal heart rate is elevated. (4) Movements of heart are diminished. (5) Total blood volume is 30-45 per cent above normal. (6) Circulation time is prolonged. (7) Per-minute output of the heart is diminished. No significant increase in stroke volume with exercise.

The patient with constrictive pericarditis of tuberculous origin

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may present the picture of heart disease with or without parallel tuberculous activity elsewhere. Generally, the imperative nature of pericardial involvement dominates the clinical scene. Impressive features are the marked venous engorgement, hepatomegaly, recurrent ascites and pleural effusion and the small paradoxical pulse. The heart is rarely enlarged, the heart sounds are faint and murmurs are absent. Blood pressure and pulse pressure are low. Mild pulmonary congestion is paled by the marked peripheral venous distention. Dyspnea at rest is rare and when present is expressive of massive pleural effusion and ascites. The patient may be febrile and this response is an important index of tuberculous activity.

The elevated venous pressure is not appreciably altered by pleural and abdominal paracentesis. Frequent venous pressure readings give reliable objective evidence of the progress of the disease. The return of venous pressure to normal is the prime criterion of successful therapy. Radiological study of the heart is an important aid in diagnosis.[8,11] Roentgenoscopic signs are: limitation of cardiac shift with changes in position of the patient, decrease in amplitude of cardiac pulsation, and limitation of the elongation of the heart with descent of the diaphragm. Roentgenographic findings are: pericardial calcification, a small aortic knob and a triangular or globular shaped heart. The x-ray kymogram gives a graphic record of the reduced excursion of the heart muscle. Opinions vary as to whether the kymogram record changes

FIGURE 1

FIGURE 2

Figure 1: Chest x-ray film preoperatively.
Figure 2: Kymogram, eight months postoperatively.

consistent with relief secured by surgery. Heuer and Stewart[11] and White[12] have described electrocardiographic changes in chronic adhesive pericarditis. These are mainly distinguished by low QRS complexes and flat T waves.

Definitive therapy is the removal of the binding pericardial-epicardial scar. All efforts are bent to eliminate edema and serous effusions in preparation for surgery. A low salt, high protein diet is given. Mercupurin and ammonium chloride are effective diuretics. Large effusions may require paracentesis. Digitalization is not recommended because it has been shown to decrease the per minute output of the heart in this condition. Burwell[3] feels that digitalis may be indicated where there is auricular fibrillation; when failure of the atrophic myocardium is expected after pericardiectomy; and in the rare case that has added myocardial failure. Quinidine may be used preoperatively, as in this case, to forestall ventricular fibrillation when the heart is being manipulated. Conflicting opinions exist as to the necessary limits of cardiac decortication. Most American surgeons advise liberating the right and left ventricles without going beyond the auriculoventricular groove. The auricles and great vessels are avoided.[9] Some European surgeons[10] are content with freeing the left ventricle. Practically, the extent of decortication is dictated by the manifold dangers of adherent coronary vessels, attenuated myocardium and obliterated cleavage planes. In addition, the surgeon must skirt the hazards of ventricular fibrillation and cardiac

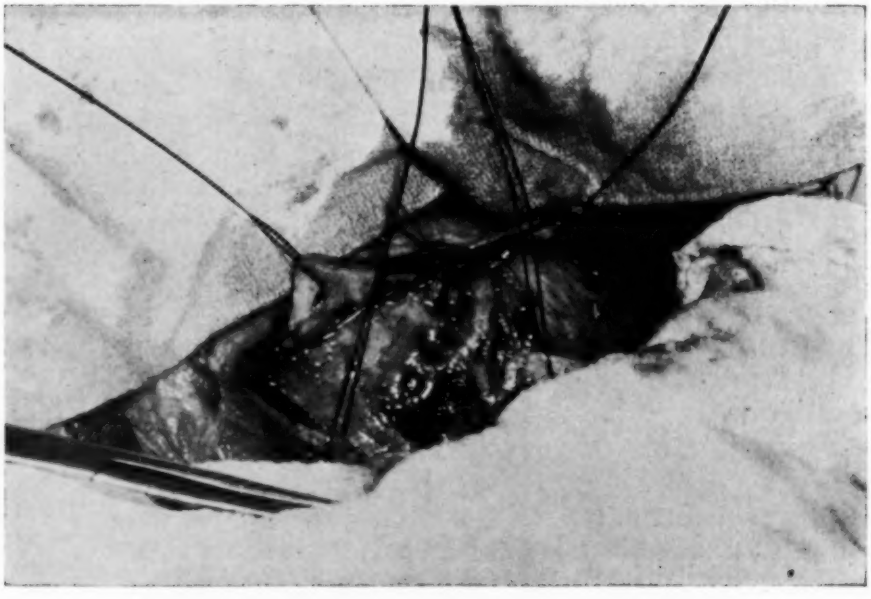

FIGURE 3: Pericardial scar at operation.

arrest. Hence, physiological needs are frequently subordinated to surgical caution. Pericardiectomy is not calculated to, nor does it affect the basic tuberculous infection. Spread and recurrence may develop after operation. Appreciation of this fact is required to explain some of the dismal results in the face of adequate surgery. Perhaps streptomycin will fill the need for controlling the element of infection.

FIGURE 4: Angiocardiogram, two and a half months postoperatively.

The determination of the optimum time for surgery requires exquisite judgment. There is general agreement that operation should be delayed until the tuberculous infection has become inactive. Certainly, morbidity statistics confirm this view. Unfortunately, the rapid march of compressive symptoms may dictate surgical intervention before infection has subsided. The surgeon must weigh the relative influence of infection and constriction, and place most emphasis on circulatory interference.

The following case is presented:

The patient is a 31 year old white barber. Familial and past history were not related to the present illness. In January, 1947 he was stricken with a pulmonary infection alleged to be pneumonia. He was treated with penicillin and remained in bed for two weeks. The patient tried to resume his job, but was forced to quit working because of increasing dyspnea and weakness. He had an unproductive cough. There was no hemoptysis. He was hospitalized at the Veterans Administration Hospital, Tuscaloosa, Alabama. At that institution, his chest was tapped and about one quart of clear yellow fluid was removed. His abdomen and ankles began to swell and he became progressively weaker. He was transferred to the Kennedy Veterans Hospital, VAMTG on March 30, 1947.

On admission the patient appeared cyanotic, dyspneic and chronically

ill. His neck veins were distended. The left hemithorax was flat to percussion. No breath sounds could be heard over the same area. The PMI of the heart could not be felt. On percussion, the heart was not enlarged. Heart sounds were distant. There was regular sinus rhythm. No murmurs could be heard. Blood pressure was 80/60. The pulse was paradoxical. Abdominal distention and shifting dullness were apparent. Hepatosplenomegaly was present. There was 2 plus pitting pretibial edema.

Laboratory Findings: Blood count, RBC 4.8 million, WBC 9,500, polymorphonuclears 78, lymphocytes 18, monocytes 4. Urinalysis: Light amber, pH 4.5, sp. gr. 1.005, albumen negative, sugar negative, many fine and coarse granular casts, occasional WBC. Blood chemistry: total protein 6.5 per cent, albumin 3.6, globulin 2.9. Serum bilirubin 2.2 mg. per cent. Malaria smear negative. No agglutination for typhoid, paratyphoid, brucella abortis, tularemia. Blood culture sterile. Urine culture, staphylococcus aureus and gamma streptococcus. Pleural fluid: protein 1.8 per cent, smear and culture negative for tuberculosis and other organisms, many WBC, no tumor cells. X-ray film of chest: moderate pleural effusion on the left. Fluoroscopy: massive left pleural effusion with displacement of the mediastinum moderately to the right. The heart was not enlarged and no pulsations of the cardiac outline were seen. Venous pressure: 32 cm. of water.

The patient was seen by Dr. Abraham Gootnick, Chief of the Cardiovascular Section who made a diagnosis of constrictive pericarditis. A tuberculous etiology was entertained, but could not be proved. The diagnosis was based on: (1) Elevated venous pressure. (2) Pleural effusion. (3) Ascites and dependent edema. (4) Low blood pressure. (5) Paradoxical pulse. (6) Absence of valvular murmurs. (7) Enlarged smooth tender liver. (8) ECG showing low voltage, and inverted T waves in all leads.

The patient was transferred to the cardiovascular service for further study and preparation for surgery. A low salt, high protein diet was given. Repeated left thoracentesis and abdominal paracentesis were performed. Mercurial diuretics were administered. The effectiveness of these measures was limited, since the pleural effusion recurred rapidly. However, ascites and peripheral edema became less marked. The patient remained afebrile until April 28, 1947 when he suddenly had several bouts of fever mounting to 104 degrees F. The fever was presumed to be evidence of the activation of the underlying pericardial disease. Gradually, the fever reached a lower level. Relief of circulatory interference, by medical means, was transitory. It became evident, that despite the active infection, surgery was indicated to release the seriously compromised heart. Quinidine was administered for four days and on May 15, partial pericardiectomy was performed.

OPERATION

Findings: The pericardium varied in thickness from 3 to 7 mm. The adhesions between the epicardium and pericardium were pliable and relatively easily separated by blunt dissection. There was a fairly definite band around the great vessels att he base of the heart. The apex was freed completely, as was the diaphragmatic surface. The pleura was very adherent on the left, and could not be stripped as completely as desired. The right auricle was partially exposed. The region of the superior vena cava was freed as much as possible from the left side but

could not be safely freed entirely. Apparently the inferior vena cava was released by this separation.

Procedure: The patient was placed on his back. The usual skin preparation was done. A curved incision was made over the second rib extending downward to the sternum, and laterally along the 5th cartilage. This was extended through skin, subcutaneous tissue, and pectoralis muscle. Portions of the 2nd, 3rd, 4th and 5th cartilage, and ribs were resected subperiosteally. Internal mammary vessels were ligated, and incision was made through the intercostals, exposing the mediastinum. The left pleura was stripped to the left. Several holes were made and repaired with atraumatic catgut. Apparently no hole was made in the right pleura in stripping it back. An incision was made over the heart through the pericardium, and a line of cleavage was established. An incision was made at right angles to the original incision, and the flaps kept intact and carried in all directions. Excess pericardium was removed, and the heart was further freed by blunt finger dissection as far as possible. The anterolateral surface of the aorta was freed, releasing a band around the base of the heart. One per cent novocaine was dropped on the heart from time to time. The space was irrigated with saline and 200,000 units of penicillin was instilled. The wound was closed with interrupted catgut sutures with two rubber tissue drains in place; one into the mediastinum, and one anterior to the periosteum and perichondrium. The skin was closed with interrupted mattress and simple sutures of silk. The patient left the operating room in about the same condition as when the operation began.

PATHOLOGY REPORT

Microscopic: Section is composed of dense hyalinized connective tissue diffusely infiltrated by mononuclear cells, epithelioid cells and occasional giant cells of the Langhan's type. Tubercle formation is widespread and most of the tubercles show caseation necrosis in their centers. In many instances the caseous necrotic areas are confluent forming larger zones in which no cellular detail can be recognized. In one section there is a rather large amount of tuberculous granulation tissue and recent hemorrhage. Near one margin the surface of both sections is covered by a fibrinous exudate. Beneath the surface the tissue is of a fibroadipose type, and contains many dilated blood vessels, neutrophiles and chronic inflammatory cells.

Diagnosis: Tuberculous pericarditis.

Immediately postoperative, there was a marked serous discharge from the wound. Cyanosis and neck vein distention receded considerably. Blood pressure was 90/70 mm. of mercury and venous pressure 15 cm. of water. On the following day the patient became icteric. Within two weeks the jaundice gradually subsided. Amigen was given to replace protein lost in the serous discharge. On the third postoperative day dyspnea recurred. X-ray inspection showed a large left pleural effusion. Drains were removed and left thoracentesis was performed. Repeated aspirations of the left hemithorax were instituted to remove the rapidly accummulating fluid. Low salt diet and mercurial diuretics were resumed. Evidence of liver damage was seen in the laboratory studies: BSP 15 per cent retention in 45 minutes, cephalin flocculation 3 plus in 4 hours, serum bilirubin 1.5 mg. per cent. Low grade fever persisted after healing of the wound. It was assumed to be due to active infection in

the remaining pericardium. The pleural fluid was a transudate and negative culture. On June 19, 1947 streptomycin therapy was begun. Firty grams of the antibiotic was given as 0.2 gram every four hours for five doses daily. The fever gradually subsided and he remained afebrile after July 30, 1947. The main difficulty was the ever recurring pleural effusion and edema. There was cause for alarm when the venous pressure rose to 22 cm. of water on August 1, 1947. An angiocardiogram showed no superior vena caval obstruction. Constriction about the caval orifice was ruled out as a cause of the venous engorgement. Kymographic studies demonstrated poor pulsations of the cardiac border. On September 26, 1947 no recurrence of pleural effusion was noted. X-ray inspection showed marked reduction in the pericardial and mediastinal thickening. The kymogram indicated increased amplitude of contractions of the left ventricle. Ascites, edema and venous distention disappeared. The enlarged spleen and liver diminished in size. Venous pressure had dropped to 7.5 cm. The patient was ambulatory and had no dyspnea on ordinary effort. On January 2, 1948 he returned from a month's leave, having gained four pounds. His liver was just barely palpable and the spleen could no longer be felt. BSP test showed no retention after 45 minutes. Cephalin flocculation was negative; prothrombin time 100 per cent; serum bilirubin 0.6 mg. per cent. The ECG exhibited a normal pattern. On fluoroscopy, increased motion of the heart was seen. The patient was considered apparently cured and transferred to a convalescent hospital February 1, 1948. Following a paranoid type of schizophrenic reaction he went home in remission July 29, 1948.

Comment

This case presented some interesting and perplexing problems. The most disturbing of these was the slow recovery of the patient. Persistent anasarca and elevated venous pressure, in the face of adequate surgery, can be a source of chargin and impatience to the surgeon. The concensus is that complete recovery can frequently not be expected for months.[1,2,6] Apparently the liberated myocardium is atrophied and requires time to regain its strength. This is a comfortable assumption if the surgeon can be assured that no recurrent or overlooked cicatricial bands compress the great vessels and the heart. Angiocardiography is suggested as a method of visualizing the patency of the superior vena cava. By such means we were convinced of innocence in that quarter. In view of the subsequent recovery it may be assumed that the patient was suffering from myocardial failure in his postoperative course.

Mercurial diuretics, salt free diet, bed rest and thoracentesis were of great value in successfully weathering this precarious period. The pre- and postoperative use of these measures must be assigned a role of importance, second only to pericardiectomy.

There is no information in the literature concerning the effect of streptomycin on tuberculous pericarditis. Streptomycin was not available, here, preoperatively. Its use postoperatively may

have curbed the activity of the disease in the remaining pericardium. Possibly, it protected the open pleural cavities from the disease. These remarks are conjectural; no unequivocal conclusions can be drawn from this case as to the value of streptomycin in active chronic productive tuberculous pericarditis. Should further study prove the specificity of streptomycin, the management of this disease will be simplified. More cases of tuberculous pericarditis with effusion may progress to healing and instances of obstructive scarring may become rare. Where decortication will be required, one can envision earlier operations on cases rendered inactive by the drug. Earlier operation will mean coping with less cicatrization and a healthier myocardium. Tuberculosis of the pericardium will not lose its terrors, but streptomycin may blunt its sting.

SUMMARY

1) The clinico-pathological aspects of constrictive pericarditis have been summarized.

2) A case of tuberculous constrictive pericarditis with partial pericardiectomy and apparent cure has been presented.

3) Freeing the heart from its fibrous encasement is the initial step in the relief of cardiac embarrassment. Postoperative supportive care of the weakened myocardium is important in the total therapy of the disease.

4) Streptomycin was used postoperatively. Its value will be determined by more extensive clinical studies.

RESUMEN

1) Se resumen los aspectos clínico-patológicos de la pericarditis constrictiva.

2) Se ha presentado un caso de pericarditis constrictiva tuberculosa con pericardiectomía parcial y aparente curación.

3) La liberación del corazón de su coraza fibrosa es el paso inicial en aliviar el embarazo cardíaco. El cuidado sustentante postoperatorio del miocardio debilitado es importante en la terapia general de la enfermedad.

4) Se usó estreptomicina después de la operación. Su valor será determinado por estudios clínicos más extensos.

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Intrathoracic Sympathoblastoma

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Intrathoracic neoplasms of sympathetic nerve origin constitute a small but exceedingly interesting group of tumors. In 1943, Wahl and Robinson[1] reported the finding of only 13 cases of intrathoracic neuroblastoma in the literature. In 1946, Hollingsworth[2] stated that the literature disclosed 16 cases of intrathoracic neuroblastoma; to these he added the report of an additional case. Although it is claimed that the incidence rate of carcinoma of the lung is increasing, there is no statistical evidence to indicate that the same applies to intrathoracic neoplasms of sympathetic nerve origin. It would seem more likely that the steadily increasing number of patients subjected to thoracic surgery has resulted in the opportunity for more accurate pathological examination of intrathoracic tumors. It can not be doubted that, in the past, patients with intrathoracic neoplasms of sympathetic nerve origin have been erroneously diagnosed, on the basis of x-ray examination alone, without subsequent histological study. We may thus expect that an increasing number of cases of intrathoracic neoplasms of the sympathetic nervous system will be reported in the future.

There has been a good deal of confusion in the classification and in the nomenclature of tumors of sympathetic nerve origin. The situation has been further complicated by the fact that the tumors frequently contain cells of different types, all of sympathetic nervous system origin, but some of undifferentiated type and others of mature cell type. One can thus appreciate the possibilities of difference in opinion and interpretation among pathologists. In regard to nomenclature, it now appears that different descriptive terms have been applied to tumors of essentially the same pathological type. In the literature there are reports of tumors variously designated as neuroblastoma, sympathoblastoma, sympathicoblastoma and neurocytoma, which apparently have been applied to neoplasms of the same histological pattern.

Clinical Aspects: In 75 per cent of cases intrathoracic sympathoblastomata are discovered in children less than ten years of age. At the time the diagnosis of an intrathoracic tumor is made, the mass is usually found to be quite large. It is thus apparent that these tumors are apt to start very early in life and one can not expect clinical symptoms to be noted until the mass

has reached such size that pressure symptoms occur or unless degenerative changes take place within the tumor mass causing cough, fever and expectoration. Hollingsworth[2] reported a case of the latter type; it is of interest that this patient was 39 years of age, and undoubtedly had the tumor asymptomatically for a considerable period of time. In the case reported below, the credit for detecting the tumor at a relatively early age should go to the parents of the child who thought they detected a slight bulge in the supraclavicular fossa when the child cried. On the basis of this observation alone, they consulted their physician whose fluoroscopic examination revealed the presence of a large mass in the left hemithorax.

In recent years mass x-ray studies have served to bring to light many instances of pulmonary pathologic changes, chiefly tuberculous lesions in varying states and stages of clinical activity. However, one must realize that the overwhelming majority of roentgen examinations of the chest have been carried out in adults. In as much as the intrathoracic sympathoblastoma is essentially a tumor of early childhood, it would be necessary to extend the system of mass x-ray study to the younger age group in order to detect these asymptomatic tumors at the earliest possible time. As the tumor is malignant in character and as the treatment is surgical, it is quite apparent that early diagnosis is imperative to achieve the optimum therapeutic results.

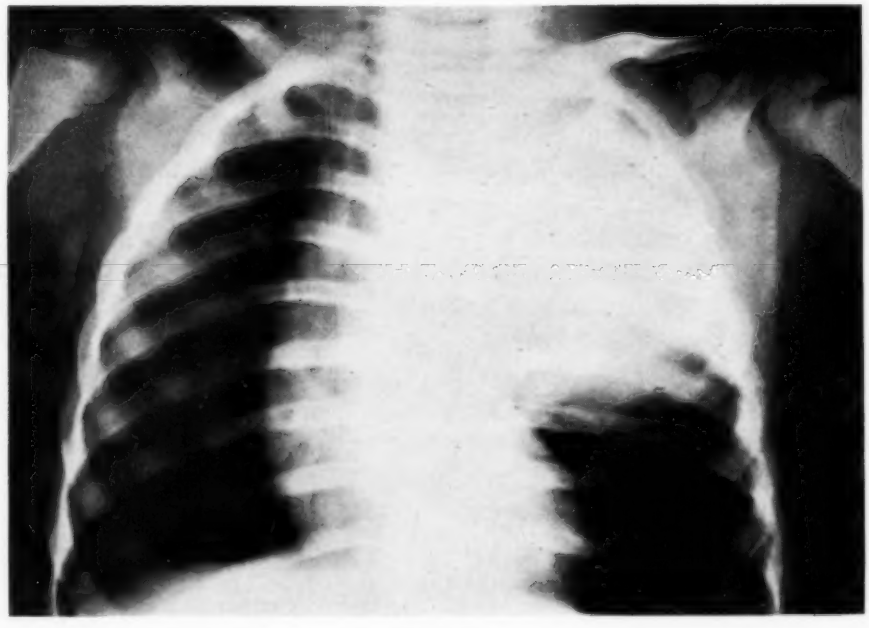

FIGURE 1

CASE REPORT

S. S., male, age 19 months was referred for examination on May 8, 1947.

History: Birth was by cesarean section. Weight at birth was 8 pounds and 3 ounces. The infant developed normally and has had no diseases. Four weeks prior to medical consultation the parents thought that they noted a slight bulge in the left supraclavicular area when the child cried. Otherwise, he seemed perfectly normal. There was no cough, hoarseness or change in voice. Temperature was normal. The child apparently had no pain or discomfort. He was examined by his physician who found a large mass in the left upper chest on fluoroscopy and referred the patient for further study.

Physical Examination: A normally developed child, 19 months of age, who appeared perfectly well. There was no inflammatory change in the nose or throat. The left chest showed a rather definite respiratory lag. On percussion there was flatness over the upper half of the left thorax. Heart and mediastinum seemed slightly displaced to the right. Breath sounds over the left upper thorax were markedly diminished. No rales were heard. Breath sounds at the left base and over the right lung were normal. Abdomen: no masses or tenderness. The spleen was not felt. There was no adenopathy. X-ray film: there was a large dense mass filling the left upper thorax. The lower border of the mass was rounded and well demarcated. The heart was not enlarged but was somewhat displaced to the right side. Diaphragm was normal in contour and in position. The right lung showed no abnormality.

Urine examination was negative. Blood study was normal. Blood chemistry: protein 6.11, calcium 10.4, phosphorous 5.5, urea 13.0.

FIGURE 2

The patient was admitted to the Brooklyn Jewish Hospital for operation. Operation, May 13, 1947 (Dr. Henry W. Louria). An oblique incision was made from the third dorsal spine to the left 7th interspace. The pleural cavity was opened and a large firm mass was found apparently arising from the posterior mediastinum. The 3rd, 4th and 5th ribs were cracked to obtain proper exposure. The blood supply of the mass was

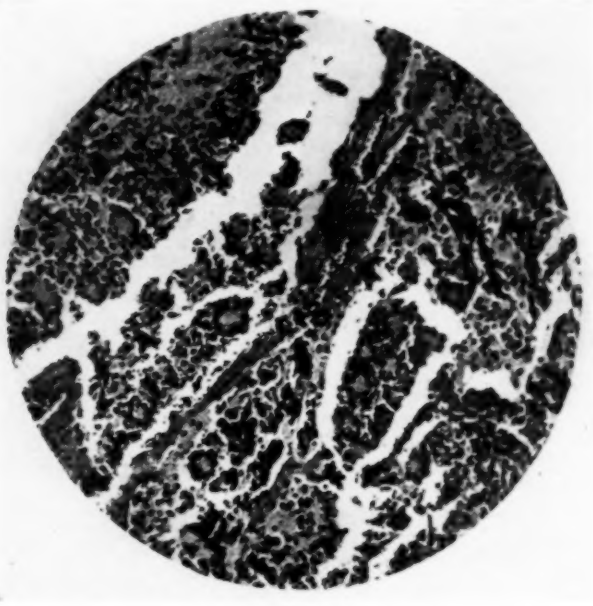

FIGURE 3

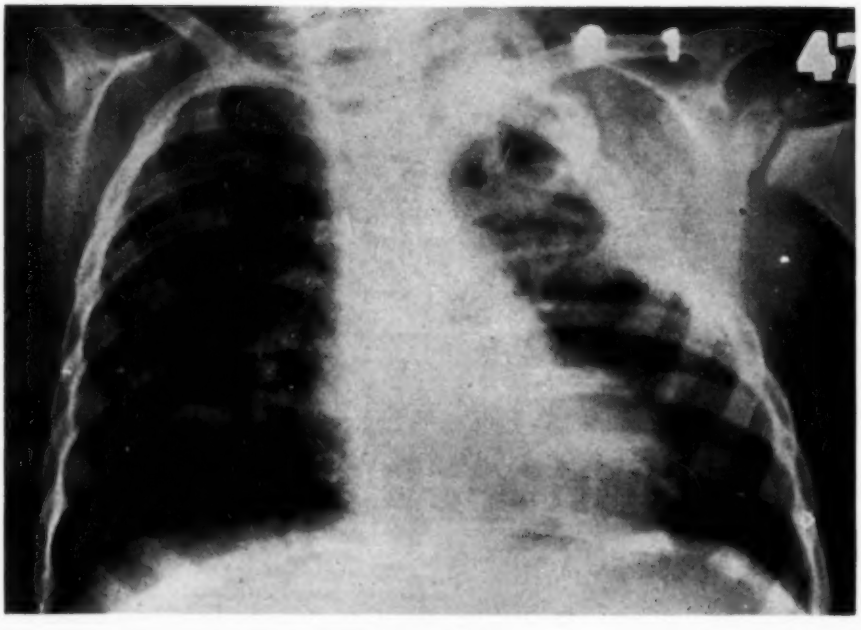

FIGURE 4

ligated and the entire mass freed and removed. A few glands were noted between the left carotid and the subclavian arteries. Closure: interrupted chromic for ribs and pleura. Skin was closed with clips.

Postoperative Course: On the first day following operation, temperature rose to 104 degrees F. and the patient had several clonic convulsive seizures. He was kept in an oxygen tent for five days. Temperature was normal on the 8th day and the patient was discharged on the 12th day after operation.

Pathological Report (Dr. David Grayzell): Specimen consists of a firm mass 9.5 x 5 x 3.5 centimeters. The external surface is covered in places by a smooth and glistening capsule which is quite firmly adherent. The remainder of the external surface is dark, red-brown in color and is roughened by fibrous tabs. The entire mass is firm and the cut surface has a central homogeneous yellow-gray area which is 4 cm. in greatest diameter. This is quite sharply demarcated from the surrounding darker tissue mass.

Microscopic Examination: The sections showed a variegated picture. In places the growth is composed of nests and sheets of large round cells with granular cytoplasm and large deep staining nuclei. In some areas no definite cytoplasm or cell outline can be made out. Elsewhere, the cytoplasm is abundant and the cells are polyhydral. Mitotic figures are numerous. Tumor cells are also seen within the lymphatic and vascular sinuses. The stroma is in places abundant, dense and collagenous and infiltrated with large and small mononuclear cells, plasma cells and polymorphonuclear leucocytes. In some areas the cells appear elongated and are deeply stained. With the Nissl stain, delicate processes are seen arising from some cells as well as granules in the cytoplasm.

Diagnosis: Neuroblastoma, mediastinal.

The child was referred for postoperative x-ray therapy. Films taken two and one-half months, and seven months after operation show no evidence of any tumor mass. The child appears perfectly well. There is no cough, fever or other symptom.

SUMMARY

A case of a clinically asymptomatic intrathoracic sympathoblastoma is reported in a child 19 months of age. The tumor was successfully removed. Eleven months after operation the child is apparently well; x-ray studies reveal no evidence of recurrence.

Routine roentgenologic studies of the younger age group will probably result in the discovery of more tumors of this type and will bring them to light at a time when surgical removal can be carried out with favorable results.

RESUMEN

Se informa sobre un caso de simpatoblastoma intratorácico, clínicamente asintomático, en un niño de 19 meses de edad. Se extirpó el tumor con buen éxito. Continúa el niño aparentemente bien

once meses después de la operación. Los estudios radiográficos no revelan signos de recidivas.

Estudios roentgenológicos de rutina de los grupos etarios más jóvenes probablemente resultarán en el descubrimiento de más tumores de este tipo y los revelarán en una época en la que es posible realizar la extirpación quirúrgica con buenos resultados.

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Bronchial Lavage in Tuberculosis

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The tuberculosis surveys we have been making since 1940 have proved once again the incompetence of radiological examination alone to affirm or deny the etiology of pulmonary disease in persons who have no expectoration and in patients with early tuberculosis. In view of this, we decided to obtain bronchial secretions directly from the bronchi through bronchoscopy. This method we had to give up on account of the annoyances and unsatisfactory results obtained with this procedure. Examination of the fasting gastric contents for tubercle bacilli was also found inaccurate and unreliable.

In 1943 we performed the first bronchial lavage using the technique we had developed for bronchographies since 1934.

Technique

The subject should be examined not sooner than from three to four hours after his last meal. He is seated on a chair in front of the physician, puts his tongue out and holds it in this position by means of a piece of gauze. He is told to breathe only through the wide open mouth. Light local anesthesia is established with a 1 per cent pontocaine solution by swabbing the tonsils, uvula and the posterior wall of the pharynx. After anesthetizing the throat, we pass through the nose a No. 14 Nelaton catheter, the tip of which will reach the oropharynx. The catheter is not introduced into the larynx or into the trachea. A 20 cc. syringe containing 5 cc. of pontocaine solution is connected to the catheter. Pontocaine is instilled drop by drop through the catheter in such a manner that it drops on the posterior wall of the pharynx. Thus it reduces the cough reflex, since we need to control it so that the patient may be able to cough at the right moment. For the anesthetic instilled through the catheter to reach the bronchus, it is essential that the patient breathes deeply through the mouth and avoid coughing.

Once the anesthetic is instilled, the patient must lie on his side, according to the location of lesion, with his head a little higher than his chest. In this position, we instill through the catheter, 15, 20 or 30 cc. of tepid, isotonic saline solution to obtain

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FIGURE 1

FIGURE 2

FIGURE 3

Figure 1, First Stage: The patient has her mouth open, breathing deeply through it, and with her tongue protruding and held by her own hand. The doctor anesthetizes her pharynx, swabbing same with a 1 per cent solution of Pantocaine. Note the position of the patient (especially the open mouth, and her tongue held with a piece of gauze).—*Figure 2, Second Stage:* The patient in the same position breathing steadily through her mouth, holding her tongue with her own hand with a piece of gauze. The Doctor passes a nelaton catheter number 14 through one nostril only until it reaches the cavum.—*Figure 3, Third Stage:* The patient in the same position, breathing steadily through the open mouth. A syringe containing 2 cc. of 1 per cent pantocaine is connected with the catheter and the anesthesia is instilled, drop by drop, by the doctor. The patient is made to breathe deeply and strongly so that the anesthesia may pass into the bronchi. This stage may be effected with the patient lying down, if desired.

expectoration. When the isotonic saline solution has been in the bronchus for 2 or 3 minutes, the patient will feel a strong urge to cough. At this moment, we give him a measure glass for sputum. He is told to cough vigorously. This expels the saline solution, together with quantities of bronchial secretions and inflammatory exudate from the lung. The material collected in the measure glass contains few Koch bacilli and for this reason, it must always be submitted to concentration method before microscopic examination, culture and guinea pig inoculation.

With this technique we have found that 90 per cent of tuberculous lesions were discharging Koch bacilli. In a number of cases with minimal lesions and with no symptoms, we have demonstrated the presence of Koch bacilli. We have carried out bronchial lavages for a great number of persons, who were living in close contact with tuberculous patients with Koch bacilli in the sputum. The former did not show shadows in the chest roentgenogram, still by the lavage, we have found that 10 per cent of them had Koch bacilli.

It is recommended to make bronchial lavage for all persons, who, with no x-ray shadows, are living together with persons

FIGURE 4

FIGURE 5

Figure 4, Fourth Stage: The patient lies down on a chaise lounge, with her head a little higher than the rest of her body. The mouth is kept open and deep breathing continued. The Doctor has charged the syringe with 20 cc. of tepid natural saline solution and instillates it slowly through the catheter to make a real bronchial lavage. At this time he warns the patient not to cough and to retain the solution in the bronchi one or two minutes.—*Figure 5, Fifth Stage:* When the solution instillated into the bronchi has increased the desire to cough until it can no longer be controlled, at once a graduated cup is offered, into which she coughs strongly in order to collect the instillated solution and the expectoration.

with tubercle bacilli in their sputum, in order to discover bronchial or tracheal lesions of tuberculous nature in time. For us, bronchial lavage constitutes the best means for establishing the activity, inactivity or cure of a tuberculous process, as well as to discover bronchial tuberculosis in those persons with normal radiographs.

All surveys or collective examinations should include search for the possible tuberculous etiology of bearers of shadows. Bronchial lavage is a valuable complement of the radiographic examination. Radiography alone occasions many errors; thus it may leave without medical care numerous cases who constitute dangerous sources of contagion to society.

SUMMARY

1) During the period of 1945-1948, 2,414 bronchial lavages were performed in Cuba. This number includes 614 bronchial lavages done by the author with his own technique. Of these, 500 cases were carried out at the Furbush Dispensary and 114 at his private office.

2) In 45.4 per cent of moderately advanced cases, considered as cured and already discharged, tubercle bacilli were still found by bronchial lavages. X-ray films in these cases showed evidence of fibrosis only.

3) We found Koch bacilli by bronchial lavage in 17 per cent of moderately advanced cases considered as cured and already discharged, with negative x-ray findings.

4) In minimal lesions without expectoration, we found 90 per cent positive with bronchial lavage.

5) Ten per cent of contact cases without x-ray evidence were found positive by bronchial lavage.

6) In a group of 90 cases of bronchial asthma, pulmonary cancer and bronchiectasis, tubercle bacilli were not found by this method.

RESUMEN

1) Durante el período de 1945 a 1948, se llevaron a cabo en Cuba 2414 lavados bronquiales. Este número incluye 614 lavados bronquiales llevados a cabo por el autor con su propia técnica. De estos, se llevaron a cabo 500 casos en el Dispensario Furbush y 114 en su oficina privada.

2) En 45.4 por ciento de casos moderadamente avanzados, que se consideraban curados y ya dados de alto, todavéa se encontraron bacilos tuberculosos mediante lavados bronquiales. Las películas radiográficas en estos casos sólo mostraron signos de fibrosis.

3) Encontramos bacilos de Koch por lavado bronquial en el 17 por ciento de casos moderadamente avanzados con hallazgos radiográficos negativos.

4) En lesiones mínimas sin expectoración, encontramos positivos al 90 por ciento con el lavado bronquial.

5) El 10 por ciento de casos de contacto sin signos radiográficos, se hallaron positivos con el lavado bronquial.

6) En un grupo de 90 casos de asma bronquial, cáncer pulmonar y bronquiectasia, no se encontraron bacilos tuberculosos con este método.

Cerebral Air Embolism Associated with Spontaneous Pneumothorax*

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A review of current and past literature reveals that the occurrence of air embolism following spontaneous pneumothorax is sufficiently rare to justify reporting. Myers et al,[1] in 1938 presented a comprehensive paper on the subject of air embolism and spontaneous pneumothorax complicating artificial therapeutic pneumothorax but cited no instance where an air embolism and spontaneous pneumothorax occurred simultaneously.

Blumberg[2] in 1940 described a case in which there was cerebral embolization as well as subcutaneous emphysema resulting from spontaneous pneumothorax. In contrast to Blumberg's patient, where the diagnosis was established primarily by autopsy, the diagnosis in this case was established by clinical and laboratory methods.

Case History and Course

The patient H. Z. is a 50 year old white male who entered the National Jewish Hospital in July 1943. The admission examination showed the presence of bilateral apical and sub-apical tuberculosis. Bilateral pneumothorax was induced with a resulting bilateral effusion six months after induction. At no time was the fluid positive for tubercle bacilli. Right pneumothorax was discontinued in August 1945 and left pneumothorax in May 1946. Hospital course was uneventful until September 4, 1947 when patient became unconscious while shaving and fell to the floor. The total period of unconsciousness was about 15 minutes. Examination, which was carried out almost immediately, showed a motor aphasia, some weakness in the right arm; there were, however, no pathological reflexes present. Blood pressure remained unaltered at 114/84. Spinal tap was negative and subsequent culture for acid-fast organisms showed no growth.

On the following day the patient was found to have a right Babinski reflex as well as sustained ankle clonus. There was some alteration of the sensory modalities in the right arm laterally and no the malar surface of the right cheek. A chest x-ray film revealed a spontaneous pneumothorax on the right with some fluid obscuring the costo-phrenic angle. The next day the patient, following a period of emotional lability in which he hurled a glass of water out of the room into the corridor,

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suddenly lost his motor aphasia. The Babinski was still present in the right foot.

On September 9th, the left pupil was found to be larger than the right. The "snout reflex" of Wartenberg, indicative of non-specific encephalopathy was positive. The Babinski was negative and the sensory modalities appeared to be intact. Fluid obtained from the right pleural space was positive for tubercle bacilli. An electroencephalogram showed no evidence of epilepsy or localized cortical pathology. At this time he began to run a toxic febrile course and fluoroscopy revealed increase of fluid in the right pleural cavity. Manometer readings were atmospheric before and after aspiration of fluid and air; thus justifying a diagnosis of patent bronchopleural fistula. Thoracotomy was performed with subsidence of toxicity. Since then, the course has been one of slow deterioration.

Discussion

The following clinical entities were entertained in the differential diagnosis to explain the neurological signs: Tuberculous meningitis, epilepsy, cerebral embolism, hemorrhage, or thrombosis and cerebral air embolism associated with spontaneous pneumothorax. Meningitis was ruled out with the absence of typical spinal fluid findings. The electroencephalogram was normal. Gibbs et al[3] in a large series of unselected epileptics found dysrhythmias in over 80 per cent of those over 20 years of age. Those under 20 years showed EEG changes in over 90 per cent of the cases. According to Busse and Ebaugh[4] better than 90 per cent of patients suffering from petit mal have disturbed electro encephalograms. In the absence of former history and the findings described, epilepsy was discarded as a plausible diagnosis. Cerebral hemorrhage and thrombosis tend to occur in individuals with hypertension or generalized arterio-sclerosis, neither of which are present in this patient. The neurological disturbances are apt to be of longer duration than in our case. It was felt therefore that this diagnosis would not account for the picture seen.

Cerebral air embolism best explains the symptom complex observed. The occurrence of spontaneous pneumothorax and cerebral accident were to co-related to be coincidental. The negative EKG, EEG, and spinal fluid all indicate that the factor causing the cerebral accident was fleeting in nature and quickly absorbed.

SUMMARY

A case of cerebral air embolism following spontaneous pneumothorax is described.

RESUMEN

Se describe un caso de embolia gaseosa cerebral consecutiva a un neumotórax espontáneo.

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Dusts of Clinical Significance

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Dusts and the diseases and disabilities they cause must always be of interest and importance to the family physician, chest surgeon, and industrial physician. The family physician usually sees the patient first; the chest surgeon on occasion may find his patient's health influenced directly or indirectly by dust; the industrial physician is concerned with prevention of any and all forms of dust diseases. All of them must meet the problems of differential diagnosis. Dust diseases as a class are contracted almost entirely from exposure during work.

Dusts are solid particles of such minute size that they can be picked up and moved easily, even by minor air currents. We may include the dusts derived from mining, crushing, stamping, grinding various rocks and ores containing silica and asbestos. We may include toxic dusts such as those containing lead, arsenic, and other inherently poisonous materials. Further there are fumes, defined as streams of very fine metal particles arising when certain metals are heated, examples being zinc oxide, copper oxide, magnesium oxide, lead, lead oxide, manganese dioxide. Other dusts, common in Texas, are sulfur, carbon black, antimony, lime stone. Likewise, we should mention those dusts, organic in origin, such as pollens, which produce allergic reactions on skin, bronchial musculature, or other parts of the body. Another organic dust is cotton, during the various phases of milling, spinning, and weaving. And finally there is the subject of dust as a vector or vehicle, dust not noxious per se but as a means of bringing infection into the body, germs which can cause havoc by riding in on the solid particles of some dust.

It is easy to believe that dust which is carried into the lungs in inspiration might cause harm in several ways, such as,

1) The fibrotic responses some of them provoke.
2) Increased susceptibility to infections such as tuberculosis.
3) Providing a vehicle for entry of pathogenic organisms.
4) The inherent toxicity of the materials,

yet the number of dusts concerned in such reactions is not great so far as known today. Certainly dusts may have significance more than is yet recognized and so the subject should be re-examined frequently. On the other hand, it is possible to arouse

fear and unnecessary anxiety by undue stress upon data too little understood.

By all odds the most important dust is silica, considered in the light of both the first two points above. As is well known, exposure to dust containing SiO_2 in particle size under 5 micra in sufficient quantity over a sufficient time can provoke chronic interstitial pneumonitis which progresses to fibrosis of a nodular character and may result in impairment of pulmonary function. We know very little about what a sufficient quantity is. The amount of time required undoubtedly varies with individuals, possibly with particle size. Degree of fibrosis from what seems to be similar exposure also is unpredictable. Another variant is the degree of dyspnoea from proportionate amounts of fibrosis.

In spite of these many doubts, it is agreed that dusts containing SiO_2 from certain sources, notably mining of hard siliceous rock (copper, gold, silver, zinc, iron, and hard coal); quarrying granite, sandstone, quartz, slate rock, and crystal; ceramic industries; and abrasives such as sand, sandstone, sandpaper, scouring material, soaps; brick making, if silica is used; stone finishing; construction of tubes, tunnels, aquaeducts; certain types of spraying jobs; all these in certain instances have resulted in silicosis to a greater or less degree of crippling. The diagnosis is made on the history of exposure, and x-ray findings of the characteristic nodular shadows. Some cases are discovered before serious reduction of vital capacity has developed. Such patients, if removed from further exposure, may live out their lives in comfort. Reduced vital capacity is not returned to normal or even lessened by any means now known. Prophylaxis by aluminum metal powder and lately by aluminum hydroxide offers some hope and in the opinion of some, these agents may be useful therapeutically though many others doubt this latter use.

One of the most serious aspects of silicosis is its association with tuberculosis. It is agreed that the presence of the two in the same individual means a prognosis poorer by far than if they are not combined. Probably the most serious part of the association of these two conditions lies in the fact that when combined the possibility of treatment and control is reduced seriously if not lost completely.

The other phases of silicosis have been covered adequately so many times that further consideration here seems unnecessary. It is still the best example of that group of diseases known as the pneumonoconioses. Let us turn to some of the dusts less well known.

First of these may well be asbestos which is a dust related because it is hydrated magnesium silicate. The hazard is en-

countered not in the use of the product but in mining, handling, and crushing the ore, and in the manufacture of various asbestos products. When inhaled in sufficient quantity, this dust produces pulmonary fibrosis which is diffuse, peribronchial, and basal at first, but may extend to all parts of the lungs as the exposure continues. Diagnosis of asbestosis in its early stages is not easy, but is made on history of exposure together with a somewhat diffuse fibrosis of lung fields, in some cases a "ground glass" appearance, with obscuring of vascular and lung markings.

Treatment is unsuccessful in reducing the involvement. Exposure should be stopped but even so there is a tendency for symptoms to become worse progressively. The only effectual control is prevention.

Another dust, common in Texas, which might be important is cotton. Yet the case against cotton is not at all clear cut. English and continental authors have been writing about byssinosis and describing chills, dyspnea, fever, and other symptoms for years. True these symptoms are reported only in those workers who are required to remain in atmospheres heavy with cotton dust over long hours, chiefly indoors. Cotton pickers for example, seem to be little affected.

When it does occur, there is dyspnea, rather asthmatic in type, a dry cough with a very little expectoration of a thick tenacious mucus. There is a tendency for these symptoms to clear on discontinuing work in cotton dust, on vacations, and even over the week-end. In fact, some of the symptoms are listed occasionally under the name of Monday fever. It is uncommon to find any evidence of fibrosis at necropsy of persons who have been exposed to cotton dust for years.

In Public Health Bulletin No. 297, issued by the Federal Government, the case against cotton is reviewed for 72 pages and 249 references are listed. It states that the "dust hazard in the cotton industry may be considered to be the most important in relations to affections of the respiratory tract." Histamine in variable quantities has been found. Several potentially harmful constituents have been noted, such as gossypol, a lipoid, and a protein substance unnamed. There is the inference that this is derived from bacteria, such as aerobacter cloacae. Insecticidal residues or disintegration products may be considered as also the high content of molds, bacteria, and spores. In general reports going back to 1818 show that illness from all causes to be commoner in cotton workers than in the general population. This is especially true of respiratory diseases. But there is progressively less differentiation as the reports become more recent, suggesting that possibly general health conditions have been improving.

Another side of the general picture, not usually considered as dust, is that of fumes. Let us hasten to add the definition which some authorities insist upon, that a fume is a stream of solid particles of extremely minute dimensions, such as arise in welding. The word is often used to mean vapors or clouds of material in a gaseous state. Such is not our meaning here. During welding such as the electric welding which was used so extensively during the war, the rod is melted by the electric current, and joins two metal surfaces. In the process, a stream of fine metal particles, actually solids, arises and often because of cramped quarters or the position of the operator, is breathed into the lungs. The effects, if any, depend upon the degree and time of exposure and upon the metals used in the rod, especially in the coating of the rod. Welder's siderosis shows metal deposits in the parenchyma and increased bronchial and hilar shadows. The importance of this lies not in mortality or morbidity, for this type of siderosis seems fairly innocuous, but rather in the danger of confusing the lung picture with more serious disease.

Perhaps one of the newest developments in the picture of dust diseases is the present interest in beryllium. The cases which have been labeled beryllium poisoning have occurred in industries which mine, grind, prepare or use beryllium in crushed or powdered form; for example, in the preparation of fluorescent screens for x-ray machines, and in the making of fluorescent lamps. The diseases associated with beryllium consist of a form of acute pneumonitis, irritation of the upper respiratory tract, skin, and eyes, and a pulmonary granulomatosis. They may be fatal, but usually are not.

Beryllium pneumonitis occurs among workmen who cut the metal on grinding wheels, and may occur in any one who has been exposed to beryllium metal or salts, especially the acid salts, in a fine dust for several weeks. It may occur during or even 2 to 3 weeks after exposure has finished. It comes on as a dyspnea, with or without fever, reduced vital capacity, substernal distress, and may have circulatory embarrassment. Many persons recover slowly. Possibly the powdered metal irritates and causes vascularization and widespread alveolar effusion, which often clears up slowly and completely. Treatment is supportive and symptomatic.

SUMMARY

Although many dusts may cause disability in isolated instances and many others may prove to be of considerable significance upon further investigation, the one dust of great and known hazards is silica. The other chemically related dust, asbestos, is known to cause great disability. On the other hand, silica in non-

crystalline or gel form, at least in commercial mixtures with aluminum hydroxide, appears to have little if any fibrogenic qualities. Here is an interesting and probably important problem, why do SiO_2 crystalline and SiO_2 gel give such different results in terms of fibrosis of the lungs?

Cotton dust is considered to be noxious but variable in its effects and, in general, the case against cotton is not clear-cut. Beryllium grinding, stamping, polishing, and crushing produces a dust which has given even fatal pneumonitis. This hazard is recently recognized, but the industry is not widespread.

Methods are available to eliminate dust exposure entirely or to a degree sufficient to avoid serious consequences. Most companies have installed or provided safety equipment. A problem still not solved is to get all workers to use the equipment consistently.

RESUMEN

Aunque muchos polvos pueden causar incapacidad en casos aislados y muchos otros podrán demostrar tener considerable significado cuando se les investigue más a fondo, el polvo de gran y conocido peligro es la sílice. El otro polvo, el asbesto, relacionado químicamente a la sílice, también se conoce que causa gran incapacidad. Por otro lado, la sílice en forma no cristalina, esto es, en forma gelatinosa, por lo menos en mezclas comerciales con hidróxido de aluminio, parece tener o pocas o ningunas cualidades fibrogenéticas. He aquí un problema interesante y probablemente importante, ¿por qué es que el SiO_2 cristalino y el SiO_2 gelatinoso dan resultados tan diferentes en cuanto a la producción de fibrosis en los pulmones?

Se considera que el polvo de algodón es nocivo pero variable en sus efectos y, en general, el pleito contra el algodón no es claro. La pulverización, trituración, abrillantamiento y molición del glucinio produce un polvo que ha causado neumonitis hasta fatal. Se ha reconocido este peligro recientemente, pero la industria no es muy extensa.

Existen métodos disponibles para eliminar la exposición al polvo por completo o hasta un grado suficiente para evitar las consecuencias graves. La mayor parte de las compañías han instalado o suministrado equipo salvo. Un problema todavía no resuelto es hacer que los trabajadores usen consistentemente el equipo.

The Use of Bronchoscopy in Bronchiectasis*

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Bronchiectasis is a serious, progressive, and widespread disease, which carries a high morbidity and mortality, and should be recognized as such. Its management depends on a sound knowledge of disease of the entire respiratory tract, and involves medical, bronchoscopic, and surgical treatment.

Bronchoscopy is a necessary adjunct in diagnosis and treatment, and although it now is occupying a diminishing role with the advances of iodized oil bronchography, chemo- and antibiotic therapy, and surgery, it still has much to offer. This is particularly true in the field of prevention, for by its use in the treatment of known predisposing causes, many cases of bronchiectasis are prevented. The increasing success of thoracic surgery in the treatment of intrathoracic diseases has no better example than the cures now possible in victims of bronchiectasis, and bronchoscopy has contributed no small part to this success.

Neither bronchoscopic nor routine roentgenographic examination will establish a diagnosis of bronchiectasis. But the information obtained from them in addition to the information obtained from iodized oil bronchograms will establish an exact diagnosis.

The appearance of the mucous membranes, as viewed endoscopically, varies widely. In most cases there is a red, swollen membrane, particularly in the bronchi serving the involved broncho-pulmonary segments; or there may be a marked acute bronchitis if the examination is done at the time of an exacerbation, so common in these patients. The membrane frequently bleeds easily, but rarely profusely, under instrumental manipulation. At times the orifices to the secondary bronchial divisions may be found occluded by an edematous membrane or by actual granulation tissue.

The amount and character of the bronchial secretions encountered during bronchoscopy may be influenced by the severity of coughing induced by the induction of topical anesthesia to the pharynx and larynx; and since the preservation of an active,

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productive cough is desirable, both for diagnostic and therapeutic reasons, tracheal and endobronchial anesthesia is not advised. Candidates for bronchoscopy will frequently expectorate such large quantities of heavy purulent material while the pharynx and larynx are being anesthetized that only small amounts will be found within the bronchial tree on examination. However, in most cases the lobar distribution of the disease can be determined by observing the individual bronchial orifices during forced coughing. This fact is extremely important in deciding which side harbors the more active infection in bilateral cases, apparently suitable for operation. Quite frequently we have encountered cases in which the bronchograms showed equal distribution of disease in each lower lobe, but on bronchoscopy the secretions were found to arise almost wholly from one side. Obviously such information is of paramount importance in selecting the proper side for initial resection. One such case is illustrated (Fig. 1).

There is one bit of information derived from bronchoscopy that has never been fully emphasized heretofore in the literature. This is the odor emitted through the bronchoscope. A large percentage of our bronchiectatic patients will not complain of any odor to their sputum or breath, and no physical examination no odor will be detected. Even a direct sniff of the sputum cup may not reveal an unpleasant odor, but with the bronchoscope in the involved lung, an unmistakable, foul odor is noticed, which signifies a necrotizing infection and is therefore of considerable diagnostic and prognostic value.

The value of inspection and biopsy of the bronchial mucosa is too often overlooked in obvious cases of bronchiectasis. The diagnosis seems so certain in many cases that one is tempted to omit a direct examination, which may disclose an unsuspected foreign body, neoplasm, or other obstruction. It is our belief that all patients with this disease should be bronchoscoped, if for no other reason than more accurately to establish a complete diagnosis. Two cases of bronchiectasis, one of unsuspected foreign body origin and one associated with carcinoma, are illustrated in Figures 2 and 3. Both of these diagnoses would have been missed had bronchoscopy been omitted.

Of particular importance in cases selected for surgical resection is bronchial anatomy. Information derived from bronchography is not complete and direct inspection is necessary. The relationship of the upper lobe orifice to the trachea and the relationship of the right middle lobe orifice to the dorsal bronchus of the lower lobe are two important aspects of surgical anatomy made by bronchoscopic inspection.

Since we feel that bronchiectasis is a disease which results from

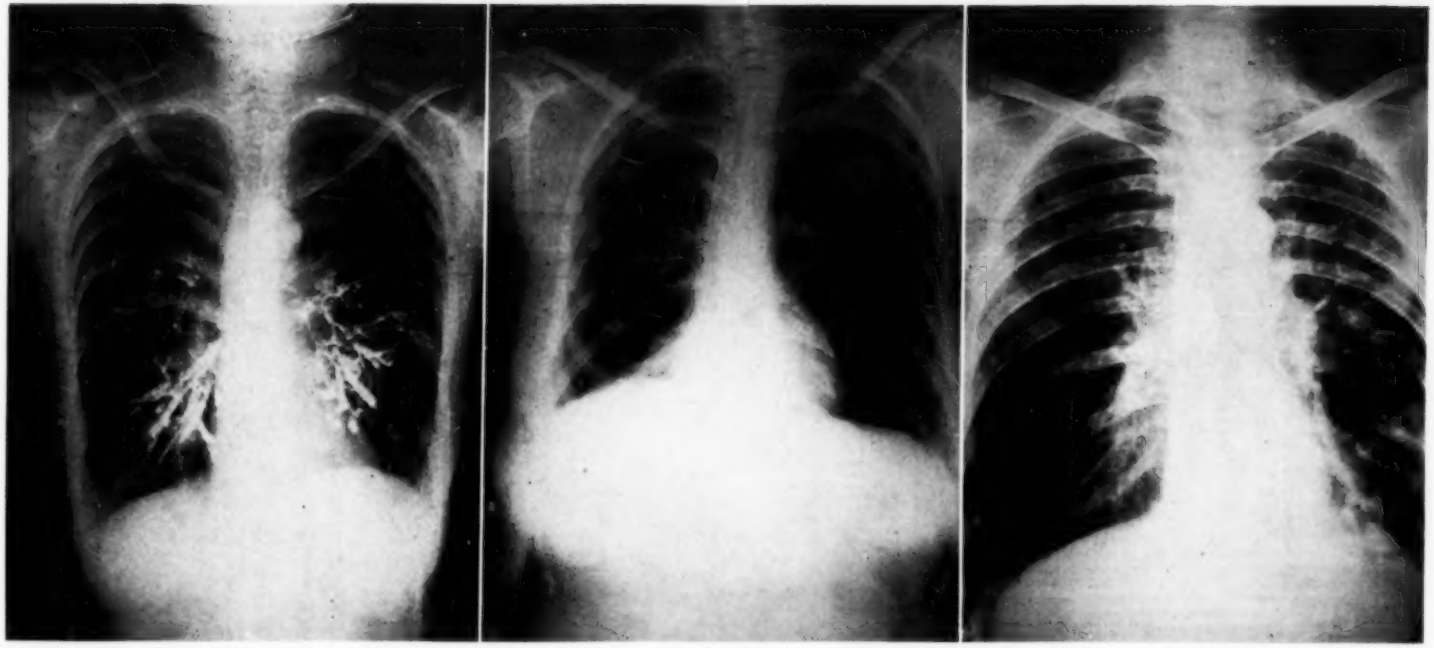

FIGURE 1 FIGURE 2 FIGURE 3

Fig. 1: A 36-year-old white female with advanced bronchiectasis of the right middle and lower lobes and the left lingular and lower lobes. The disease was equally distributed by x-ray and physical findings, yet bronchoscopy revealed the right side to harbor the most active infection. A right middle and lower lobectomy was performed with marked relief of symptoms. Resection of the disease on the left has been delayed because of many factors.—*Fig. 2:* A 40-year-old white female admitted in July 1947, with a history of having aspirated a piece of nut shell in 1938. She had cough and malodorous expectoration ever since. The films reveal a small, contracted right lower lobe. Bronchoscopy revealed a foreign body in the orifice of the right lower lobe, and after extraction, it proved to be a large piece of nut shell. After nine years the finding of the foreign body was unexpected. The patient will need a lobectomy because of the irreversible changes in this lobe.—*Fig. 3:* A 65-year-old white male with a four-year history of cough, hemoptyses, and profuse foul sputum. The x-ray film shows characteristic changes of advanced bronchiectasis and pneumonitis. Bronchoscopy revealed an extensive bronchogenic carcinoma almost totally occluding the left main stem bronchus, flush with the carina.

bronchial stenosis and infection, the bronchoscope is necessary in order to rule out or relieve bronchial stenosis, whatever its cause may be. Among these causes are: retained secretions as seen in the new-born or following general anesthesia; or inflammatory processes in the lung and bronchi; or foreign bodies, tumors, granulomas, and strictures; or compression stenoses from extrabronchial causes. Of course, in the majority of cases coming to bronchoscopy, the bronchial stenosis no longer exists.

The bronchoscope is also valuable in obtaining secretions for direct smears and cultures uncontaminated by pharyngeal and mouth bacteria. These specimens should be studied both aerobically and anaerobically for bacterial types and fungi. We have found tubercle bacilli on smears in patients in whom a diagnosis of tuberculosis could not be made otherwise. The cultures are useful for the preparation of autogenous vaccines and for determining bacterial type and resistance behavior, so that the most suitable drug may be employed. It seems logical to us that the necrotizing toxin of certain strains of staphylococci must play an important role in the production of this disease.

Finally, the bronchoscope may be useful in determining the lobar source of hemorrhage, and for making iodized oil bronchograms in certain areas which could not be filled and demonstrated by routine methods.

Bronchoscopy in Treatment

In general, bronchoscopic intervention plays an important role in the treatment of bronchiectasis in three types of cases: (1) patients with acute exacerbations of old, usually quiescent bronchiectasis; (2) patients whose disease is anatomically suitable for operation but whose poor general condition precludes operation until the infection has been brought under control and their general condition improved; and (3) patients with a hopeless bronchiectasis, such as those who are too old, or those who have other serious diseases, or those in whom the extent of the involvement contraindicates operation. In these patients bronchoscopy is then employed as a paliative procedure.

The most useful therapeutic purpose of the bronchoscope rests in the ability to aspirate bronchial secretions and to relieve bronchial obstruction. The objective and subjective relief afforded by bronchoscopic aspiration defies adequate explanation in patients who do not have a real bronchial block. It seems unlikely that a patient whose sputum is profuse and is particularly influenced by posture would gain relief for as much as several weeks from such an aspiration, but many case histories will bear out proof of this by the spontaneous return visits of these patients

requesting bronchoscopies. This relief is no doubt brought about by the violent coughing induced by the bronchoscope which causes a much more thorough expulsion of bronchial secretions than would ever be obtained voluntarily. It is easy to understand the relief afforded by release of an acute inflammatory (mucosal edema or granulation tissue) obstruction with a removal of tenacious secretions from the depths of the smaller bronchi. The actual bronchoscopic technique involves the passage of an appropriate aspirating tip, olive-tipped bougies or small bronchoscopic sponges on a carrier. Following such manipulation, the use of expectorants, inhalations, and postural drainage should be employed.

The value of the bronchoscope in permitting the application of drugs to the mucous membrane has been minimized by some and overemphasized by others. Epinephrine and other sympatheticomimetic drugs applied to a swollen membrane do have some shrinking effect, although much less so than when applied to the nasal mucosa. This action is obviously advantageous in enlarging the bronchial airway. The shrinking agent can be applied on a bronchoscopic sponge or it can be flushed into the bronchial tree directly through the bronchoscope.

The advent of the chemotherapeutic and antibiotic agents brought a brief flurry of hope that topical application through the bronchoscope would be effective. However, we now feel that our hopes were unfounded, for no demonstrable improvement was obtained, probably because of the brief period of contact of the drug. The action of the ciliated epithelium and the cough associated with bronchoscopy soon rid the bronchi of either powder or solution. The aerosol method of inhalation of antibiotics is more sound and has given better results.

Bronchoscopy is not without hazards. Occasionally a severe pulmonary hemorrhage will occur during bronchoscopy. In this event the examination should be terminated, for in our experience bleeding has ceased spontaneously after the withdrawal of the bronchoscope. Of course, a blood transfusion should be given if necessary. An additional hazard is the occasional case of spontaneous pneumothorax which may occur. Sudden chest pain and dyspnoea should make one suspect this complication.

Bronchoscopy in Prophylaxis

A discussion of bronchoscopy in the treatment of bronchiectasis must emphasize the prophylactic treatment. It is a most uncommon pulmonary infection that does not produce some secretion. This secretion is always a potential hazard because if not evacuated promptly, it will occlude the airway. Such occlusion

results in atelectasis, and however transient and however small the bronchus, irreperable damage may occur. It takes a very brief time for the combination of infection and obstruction to destroy bronchial walls, or necrotize pulmonary tissue. Opinion today favors this etiology of bronchiectasis, particularly in children. Therefore, it is urged that all cases with pneumonia, tuberculosis, tracheobronchitis, pulmonary suppuration, or any of the rarer infections be bronchoscoped without delay when the clinical course, physical findings, or roentgenographic details point to retained bronchial secretions. The masking effect of the sulfonamides and antibiotics on pulmonary infections is well recognized and should not be overlooked.

Of particular value in the technique of bronchoscopy in such a case is the use of bronchial lavage in the diseased lobe. An adrenalin solution or a solution of normal saline flushed into such a lobe will initiate coughing, which will usually result in the evacuation of secretions not visible through the bronchoscope.

The bronchoscopist can do much in his own hospital by instructing the residents, anesthetists, and nurses on the importance of (1) not depressing the cough reflex by large doses of morphia or codeine; (2) aspiration of the trachea after general anesthesia when indicated, and resorting to the inhalation of a carbon dioxide - oxygen mixture; (3) frequent turning of the patient after anesthesia and getting him up as soon as possible; (4) using postural drainage properly; (5) the value of aerosol therapy; (6) being on guard for the masking effect of the sulfonamides and antibiotics; and (7) the employment of bronchoscopy early in persistent atelectasis, particularly when infection is present.

SUMMARY

Although bronchoscopy is playing a diminishing role in bronchiectasis, its many uses in diagnosis, treatment, and particularly prophylaxis are so necessary and important that we feel that it should be employed in all cases—as a routine examination—in order to make a more exact diagnosis, as a method of treatment in selected cases, and finally as a most important and valuable prophylactic procedure.

RESUMEN

Aunque se está empleando menos la broncoscopia en la bronquiectasia, sus muchos usos en el diagnóstico, el tratamiento y, particularmente, la profilaxia son tan necesarios e importantes que opinamos que se debe emplear en todos los casos—como examen de rutina—a fin de hacer un diagnóstico más exacto, como tratamiento en casos seleccionados y, finalmente, como procedimiento profiláctico importante y valioso.

Tomography of Larynx in Disseminated Pulmonary Tuberculosis

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Since Dr. Felix Leborgne of Montevideo, in 1936, introduced tomography for the diagnostic study of carcinoma of the larynx, this procedure has become definitely established in the diagnosis of this condition. Some years before him, in 1923, Dr. Henry Courtard had emphasized the importance of a radiographic examination of this organ.

I have had opportunity to undertake tomographical studies in patients with advanced pulmonary tuberculosis who also had laryngeal manifestations visible on simple inspection. I have studied 10 cases of this nature and have found that it is possible to demonstrate with tomography changes caused by the disease, whether they are in the form of a simple inflamation or other types of specific damage. In almost all of the patients there has been agreement between tomographic findings and those noted on standard laryngoscopic examination.

As a note of technical character we are able to point out the following as for the lateral radiography, imprescendible to perform it for the due study, as for the tomography.

Technique

1) *For Lateral Radiography of the Larynx:*

200 milli. Ampers
1/20 second or 10 milli. Amper seconds
55 kilo volts for 10 to 13 cm. of neck thickness
160 cm. distance
Rotating anode tube
Focusing on the thyroid prominence
Vocalization in "U".

2) *For Tomography:*

20 milli. Ampers
3 seconds or 60 milli. Amper seconds
75 kilo volts or less
Focusing on the thyroid prominence
Vocalization in "U"
Rotating anode tube.

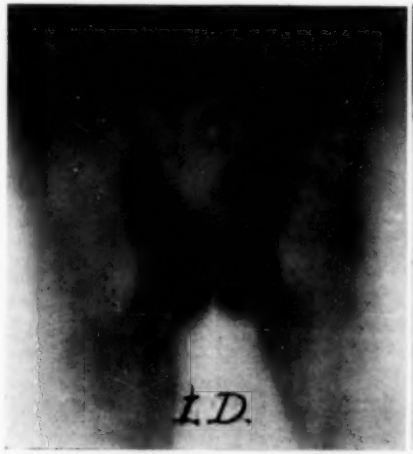

FIGURE 1

FIGURE 2

FIGURE 3

FIGURE 4

Figure 1, I. D.: Bilateral extensive caseous tuberculosis, with third degree dissemination on both sides. Occlusion of both ventricles. Laryngoscopic diagnosis: Polypoid granulation, right vocal cord.—*Figure 2, B. C.:* Bilateral extensive caseous tuberculosis with 3rd grade dissemination. Occlusion of the left ventricle. Laryngoscopic diagnosis: Arytenoidal infiltration (left) and Paresis of the respective vocal cord.—*Figure 3, W. G.:* Bilateral extensive caseous tuberculosis, disseminated. Occlusion of both ventricles.—*Figure 4, M. J. B.:* Bilateral extensive caseous tuberculosis, disseminated. Occlusion of both ventricles, with impossibility to recognize their outline or the vocal cords. Laryngoscopic diagnosis: Tuberculous laryngitis, infiltrative-edematous form.

It is necessary to emphasize the following facts: Dr. Leborngne in his book "Cancer of the Larynx," recommends that the films be taken with the patient in the supine position. In this position it has not been possible for us to get satisfactory films. On the other hand, placing the patient in the prone position, excellent results have been possible as can be seen in the illustrations.

A constant tomographic sign in all cases is the subglottic angle, which, as a rule, is rectangular, becomes frankly obtuse, regardless of the degree of tuberculous laryngitis (cases 2 and 3, arrows).

SUMMARY

This preliminary communication has been written with the purpose of calling attention to a diagnostic approach. Conclusions as to the clinical value of this method will be reported as soon as observations are gathered on a larger number of patients.

RESUMEN

Se ha escrito esta comunicación preliminar con el propósito de llamar la atención sobre un método diagnóstico. Se publicarán conclusiones sobre el valor clínico de este método tan pronto como se acopien observaciones en un número mayor de pacientes.

A Case of Fatal Tularemic Pneumonia with Necropsy*

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The following case of tularemic pneumonia, with necropsy findings, is presented to demonstrate that earlier awareness of such a diagnostic possibility might well have resulted in the patient's recovery. P. tularensis should always be considered a possible etiologic agent in severe primary atypical pneumonia.

According to the United States Public Health Service, the yearly reported incidence of tularemia in the United States has varied between 960 and 2291 for the ten year period from 1937 through 1947, with a mortality of about 5 per cent.

Clinically, tularemia has been classified as ulcero-glandular, oculo-glandular, and typhoidal. In the typhoidal type there is neither demonstrable primary lesion of skin or eye, nor superficial lymph node enlargement. Of 1856 cases reported by Francis,[1] 119 or 6.4 per cent were typhoidal.

Pulmonary and pleural involvement occur in all three varieties of tularemia, but predominate in the typhoidal. Of Foshay's[2] 554 cases of ulcero-glandular and oculo-glandular tularemia, approximately 15 per cent had evidence of pneumonia as contrasted with 52 per cent of his 46 typhoidal cases.

In a recent review of cases of tularemic pneumonia, reported in the American literature, Stuart and Pullen[3] could only collect 253, to which they added 15 of their own. Of these 268 cases, 97 or 36.2 per cent conformed to the typhoidal type of infection. Hunt[4] has added 12 and Morgan[5] 15, making a total of 295 cases of pulmonary tularemia reported to date. A diagnosis of pneumonia, primary, atypical, etiology unknown, has probably masked others.

Blackford and Casey,[6] writing before the introduction of streptomycin, reported the mortality in tularemic pneumonia to be approximately 30 per cent. Hunt[4] and Morgan[5] had but one death in 27 cases treated with streptomycin. Once pulmonary involvement occurs, tularemia becomes a real threat to life in the absence of prompt and adequate streptomycin therapy.

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Report of Case with Necropsy

A 48 year old, white, male insurance salesman, a resident of Lansdale, Pa., was admitted to Hahnemann Hospital at 8:45 P.M. on November 13, 1947.

Because of the patient's stuporous condition, the history was obtained from his wife. The patient had felt well until November 2, 1947, when he had a distinct chill, with sudden onset of severe frontal headache, fever, and dyspnea. Vomiting occurred on November 4th. By November 11th severe cough was present, and blood-tinged sputum was noted the next day. Dyspnea and fever of 100 to 103 degrees F. were prominent and persistent throughout the illness, the dyspnea becoming severe by November 11th.

He was treated by two physicians at home before hospitalization was advised, and had received a sulfonamide (type and dosage unknown) and approximately 2,500,000 units of penicillin. White counts taken on November 11th and 13th were 7,200 and 4,500 respectively, with an essentially normal differential count. The Widal test was negative.

At the time of admission to the hospital, the patient appeared critically ill, dehydrated, and was unable to answer questions. The temperature was 102.8 degrees F., pulse 120, and respirations 56 per minute, abdominal in character. The blood pressure was 134/78. Fine, medium, and coarse rales were heard from the apex to base of both lungs. The percussion note over the left lower lobe was flat, and the right lower lobe was dull on percussion. Examination of the abdomen was unsatisfactory because of marked rigidity. Bilateral ankle clonus was present. In other respects physical examination was not remarkable.

The blood count showed 10.3 grams of hemoglobin, 3,640,000 red cells,

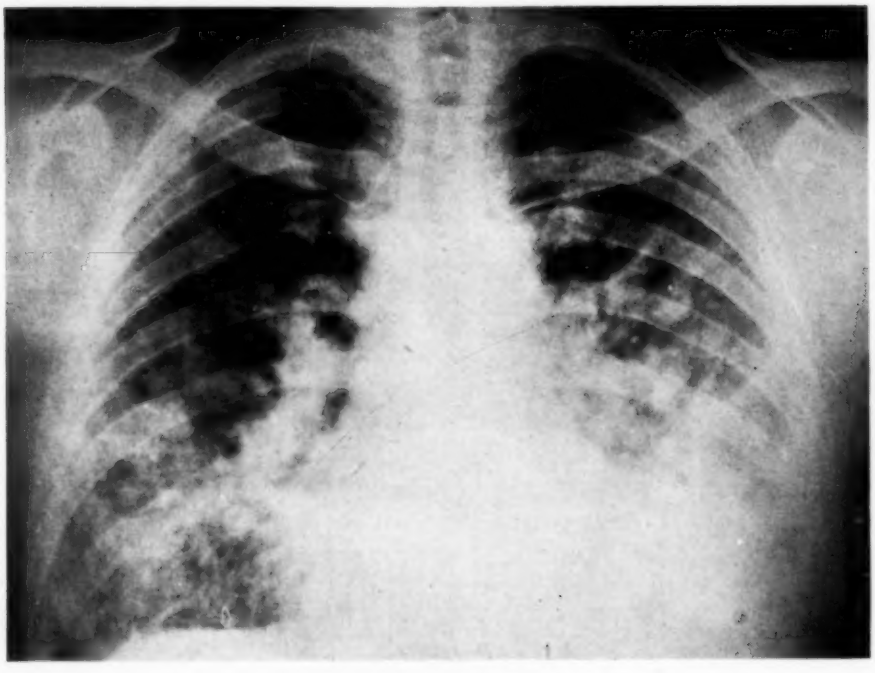

FIGURE 1: See Text.

and 4,600 white cells, non-segmented polys. 10 per cent, segmented polys. 74 per cent, lymphocytes 15 per cent, monocytes 1 per cent. The spinal fluid examination showed normal manometrics, and had a normal cell count, chloride, protein and sugar content. The blood and spinal fluid Wassermann test were negative. Agglutination tests for typhoid, paratyphoid, rickettsia and brucella abortus were negative, as were cultures of the throat, spinal fluid and blood. Examination of the sputum failed to reveal tubercle bacilli, and a culture showed a few colonies of B. pyocyaneus and pneumococci. The feces were negative for ova and parasites. Urinalysis was normal. The icterus index was 33.3.

X-ray films of the lungs revealed a patchy pneumonia involving the lower two-thirds of both lung fields, confluent at the bases, and worse on the left side (Fig. 1).

Although adequate supportive treatment was instituted in the form of oxygen inhalations and intravenous fluids, the patient's condition in the hospital steadily deteriorated. On admission full dosage of penicillin and sulfadiazine was begun, and on the second day streptomycin was given, 0.5 grams every 6 hours. Stupor deepened, incontinence of urine and feces developed, and jaundice became evident. The patient died at 4:45 P.M. on November 15th, 56 hours after admission.

The clinical diagnosis was pneumonia, primary, atypical, etiology unknown. Viral pneumonia of known etiology (influenza, psittacosis, lym-

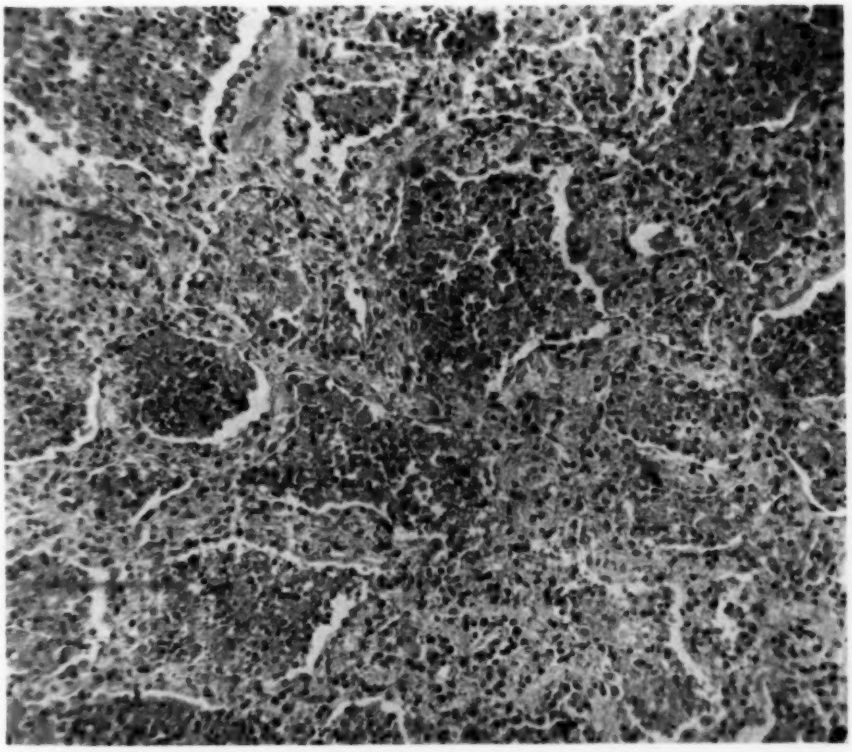

FIGURE 2: Low Power View of Necrotizing Pneumonia. Note the partially necrotic exudate filling the alveoli, the thickened alveolar walls due to edema, endothelial swelling, cellular infiltration and septal cell proliferation.

phogranuloma venereum, lymphocytic choriomeningitis) was considered a possibility, while typhoidal tularemia with pneumonia was ignored.

The pertinent findings at the postmortem examination by Dr. John E. Gregory follow. The skin and superficial lymph nodes were normal. Both pleural sacs were obliterated by dense adhesions. The tracheobronchial tree was filled with dark, brownish mucoid secretion, following the removal of which there was seen a reddened, hemorrhagic mucosa. The lower lobes of both lungs were the site of a confluent lobular pneumonia, which on microscopic examination showed a necrotizing inflammatory reaction (Fig. 2 and 3). The spleen was eight times it's normal size. Focal necrosis was demonstrated in the liver, spleen, kidneys and bone marrow. An agglutination test of heart blood for P. tularensis was negative, and cultures of heart blood were sterile. A guinea pig inoculated with bronchial secretion died shortly after inoculation, and too soon for the development of lesions. Giemsa stained lung sections showed several clusters of very small coccoid bacilli that could well have been P. tularensis. Permission to examine the brain was not granted.

The gross findings were such that further inquiry from the wife disclosed for the first time the vital information that the deceased had been hunting rabbits on November 1st, and had skinned two rabbits on the same day, 17 hours before the onset of symptoms. In view of the history, the necrotizing lesions, and the demonstration of small coccoid bacilli in the lung sections, there seems little doubt that this is a case of tularemia of the typhoidal type with pneumonia.

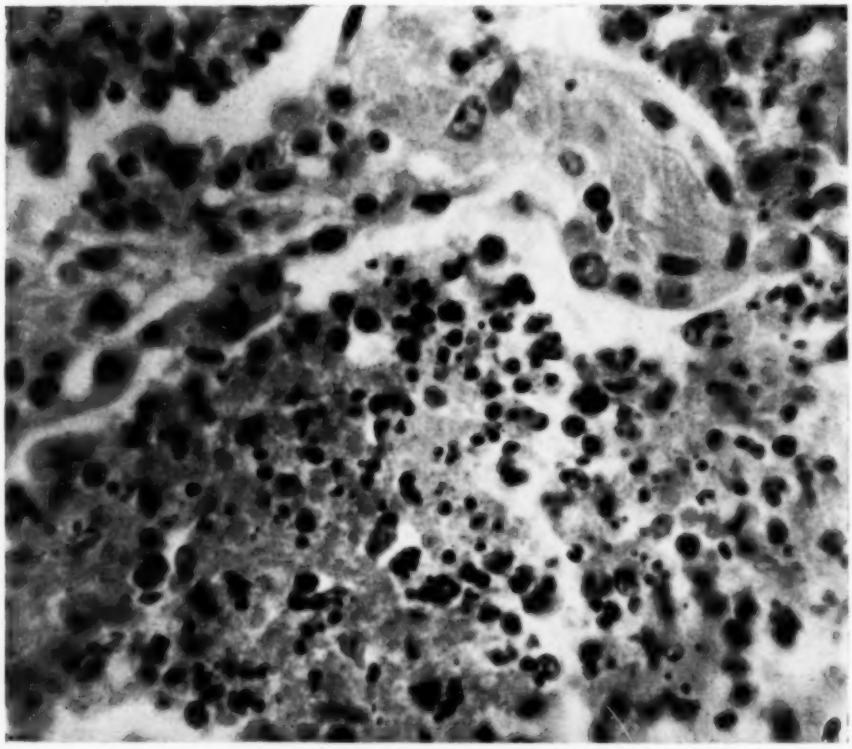

FIGURE 3: High Power of Same Field. This shows the character of the exudate and of the alveolar wall changes in detail.

Comment

Our unfortunate experience in this case has caused us to examine critically our current methods used in searching for the possible causative agents in severe cases of primary atypical pneumonia. It is well known that the isolation of P. tularensis is difficult, requiring special culture media such as dextrose-cystine-blood agar[7] and hazardous animal inoculation. Nevertheless, these procedures offer the only means of early diagnosis in typhoidal tularemia, since, as in this case, agglutination tests for P. tularensis are negative until the end of the second to fourth week of the disease. Lung puncture should be considered in the absence of sputum.

If typhoidal tularemia is recognized as a diagnostic possibility, a history of exposure to wild rabbits or ticks will often be obtained.

It would seem to us safest, as has been recommended by Hunt[4] and Morgan,[5] to give streptomycin a therapeutic trial in hospitalized cases of severe atypical pneumonia, while every available means is being used to establish an etiologic diagnosis.

Had direct inquiry been made about exposure to wild rabbits, had more careful bacteriologic investigation been made, and had streptomycin been administered earlier, the outcome in the case described might well have been favorable.

SUMMARY

1) About 6 per cent of tularemia are typhoidal, and probably half of these have pulmonary involvement.

2) A case of tularemic pneumonia, diagnosed at autopsy, is described.

3) Early inquiry as to possible wild rabbit or tick contacts should be made in all cases of primary atypical pneumonia.

4) In severe cases of primary atypical pneumonia special laboratory technics should be used in an attempt to demonstrate P. tularense, since agglutination tests may remain negative until the fourth week of the disease.

5) In such severe cases a trial of streptomycin is suggested while search for an etiological agent is in process.

RESUMEN

1) Aproximadamente el 6 por ciento de los casos de tularemia son de tipo tifoideo y probablemente la mitad de ellos tienen lesiones pulmonares.

2) Se describe un caso de neumonía tularémica diagnosticada por la autopsia.

3) En todo caso de neumonía atípica primaria se debe inves-

tigar prontamente la posibilidad del contacto con conejos salvajes o garrapatas.

4) En casos graves de neumonía atípica primaria deben emplearse técnicas especiales de laboratorio para tratar de demostrar la P. tularense, pues las pruebas de aglutinación pueden continuar negativas hasta la cuarta semana de la enfermedad.

5) Se sugiere que se ensaye la estreptomicina en esos casos graves, mientras se prosigue la busca del agente etiológico.

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Bronchographic Observations in Collapsed Lungs*

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General Considerations: The similarity between the phenomena that govern pulmonary ventilation and the entrance of lipiodol from the trachea to the alveolus, has made us think that it may be possible to record bronchographically some of the changes caused by the collapse of the lung. In fact, the lungs may be ventilated when their bronchi are patent and their thoraco-alveolar system is unchanged; likewise, the opaque material fills the major bronchi when they are patent, and the alveolus only when it is aspirated on inspiration. Stating the question in this manner, we shall not pretend to draw conclusions, but from the accompanying bronchograms we shall make some deductions whose validity we leave to the reader.

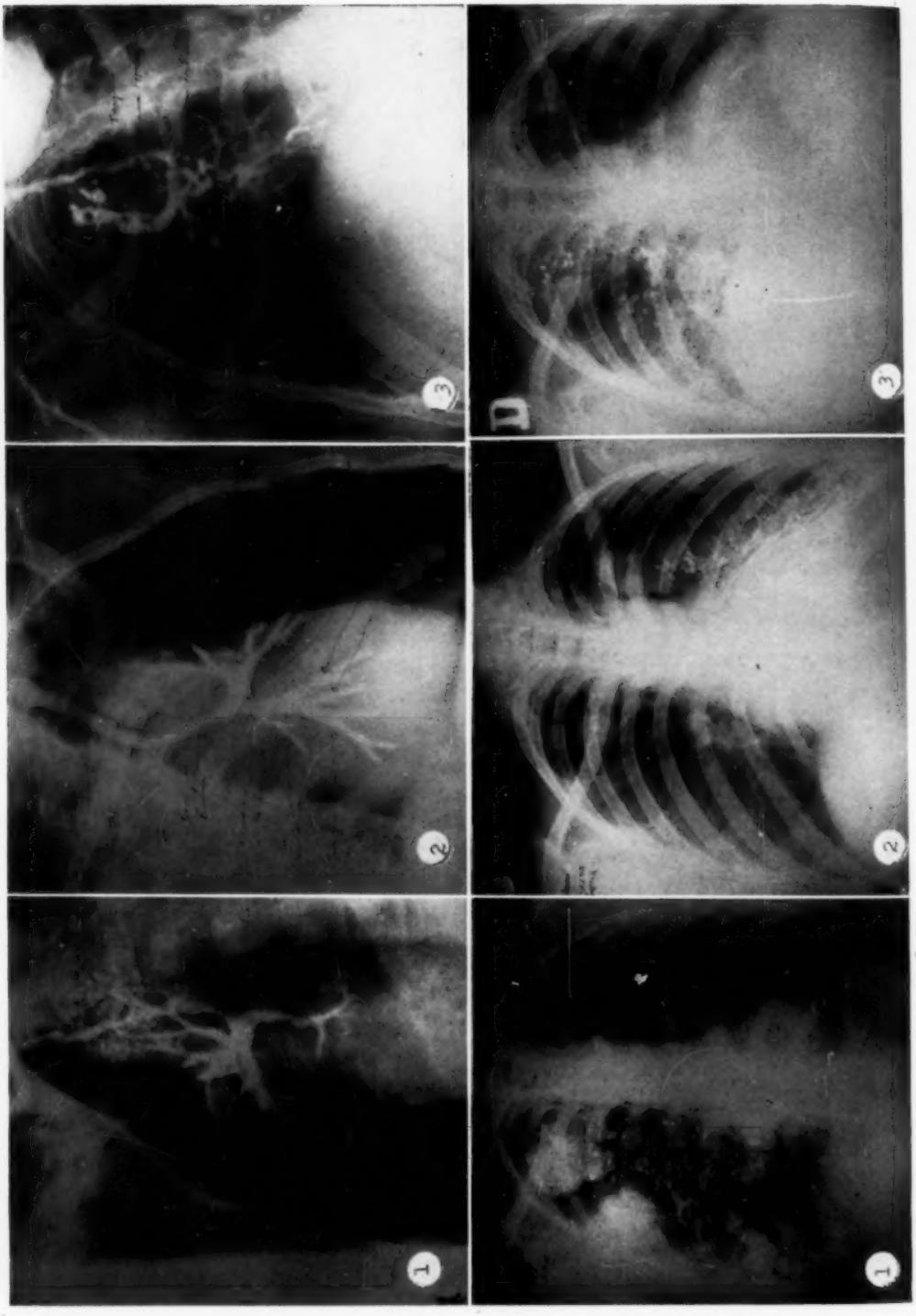
The work of S. Di Rienzo, "Radiological Exploration of the Bronchus," has served us as the fundamental basis for this study and, in addition, we have had the excellent collaboration of the radiologist Dr. Vicente Ubillús.

Bronchographic Observations: Bronchograms 1 and 2 represent normal bronchograms. Note the filling of the major bronchi of the upper lobe of the right lung and the beginning of the foliage-like alveolar filling (Fig. 1). In the film taken 10 minutes later (Fig. 2) one sees that the canalicular phase has ended and the lipiodol is found in the alveoli, appearing as masses of greater or lesser density caused by the superimposition of shadows.

Bronchograms 3 and 4: Female patient treated with left pneumothorax for eight months. Note the filling of the major bronchi of the entire left lung, *the absence of secondary branches and of "foliage,"* in spite of having kept the patient in an adequate position for 10 minutes (Fig. 3). The film taken 15 minutes later (Fig. 4) shows the abnormal persistence of the canalicular phase, the absence of "foliage," the displacement of the major bronchial branches and the air space produced by the artificial pneumothorax.

Bronchograms 5 and 6: Female patient who had a right phrenic exeresis and has been under treatment for one year. Note that in this case the secondary bronchial branches and the "foliage"

*Presented at a scientific session, November 7, 1946.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are absent in the upper lobe; the major branches are filled with opaque substance but are dilated into small sacs (Fig. 5). Fluoroscopically, it was possible to demonstrate a marked change in bronchial dynamics and tonicity. In Figure 6 one may see the elevated right hemidiaphragm and the formation of "foliage" in relation with the anterior branches of the lower lobe—this was confirmed in a lateral film.

DEDUCTIONS

1) It is evident that pulmonary collapse brings about a state of hypoventilation in the collapsed lung, due to changes in bronchial patency and in the respiratory function of the thoraco-alveolar system. These changes, demonstrable by bronchography, would be manifested in spasms and partial or total bronchial obstructions with their immediate consequences, alveolar emphysema or atelectasis, which may affect from small areas to an entire lung.

2) Phrenic exeresis produces an effect similar to artificial pneumothorax, especially in the upper lobe and the posterior part of the lower lobe; but, in addition, it causes dilatation of the major bronchi due to relaxation of their muscular layers.

DEDUCCIONES

1) Es evidente que el colapso pulmonar origina un estado de hipoventilación en el pulmón colapsado, mediante alteraciones en la permeabilidad bronquial y el funcionamiento aspirativo del sistema tóraco-alveolar. Estas alteraciones demostrables con la broncografía, se manifestarían mediante espasmos, obstrucciones parciales o totales de bronquios con sus consecuencias inmediatas: insuflaciones alveolares o atelectasia que pueden afectar desde pequeñas zonas hasta la totalidad de un pulmón.

2) La frénico-exéresis produciría acciones semejantes al neumotórax artificial, especialmente en el lóbulo superior y parte posterior del lóbulo inferior; pero además se producirían dilataciones de los bronquios principales por relajamiento de sus capas musculares.

FIGURE 1 (Top): Normal bronchogram. Initial (Canalicular) phase. (Bottom): Normal bronchogram. Formation of "Foliage.' After 10 minutes.—FIGURE 2 (Top): Bronchogram with Pneumothorax. Canalicular phase. (Bottom): Bronchogram with Pneumothorax. Absence of "Foliage." Persistence of the canalicular phase after 20 minutes.—FIGURE 3 (Top): Bronchogram with Phrenicectomy. Canalicular phase. (Bottom): Bronchogram with Phrenicectomy. After 20 min.

A Brief Analysis of Fifty Foreign Bodies in the Larynx, Trachea and Bronchi*

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The special methods of diagnosis and treatment applied in the present series of foreign bodies located in the larynx, trachea and bronchi are similar to the methods I have learned from my esteemed teachers, Professors Chevalier Jackson and Chevalier L. Jackson. Therefore, my statistical observations cannot but be a humble addition to those extensively charted, developed and described by them in 1945. At that time they had analyzed 3,122 cases of foreign bodies in the upper food and lower air passages of a total numbered collection of 4,259 to which I had the great privilege of contributing in a small way when I had the good fortune to be their assistant more than ten years ago. At present, I have a collection of 285 specimens of foreign bodies of which 80 were removed from the lower respiratory passages during the past 9 years. All the others were found in the upper food passages.

The present statistics deal with the first series, fully charted, of 218 foreign bodies. Speaking of this series, 22 per cent or 50 were found in the lower respiratory track. Of these 50, 4 were located in the larynx (8 per cent); 4 in the trachea (8 per cent); 42 in the bronchi or 84 per cent of the grand total of inhaled foreign bodies. That is to say that when a foreign body was inhaled, in 85 per cent of the cases it was aspirated into a bronchus, and two times out of three into a right bronchus.

The nature of the foreign bodies was rather varied: 22 peanuts or 44 per cent of the total; 10 other vegetable or organic bodies (20 per cent); 16 metallic bodies (32 per cent). Forty-four of these patients were children (88 per cent); 6 only were adults (12 per cent). This difference can be explained by the tendency on the part of children to bring every object to the mouth and to place small objects in the mouth. One of our women had inhaled her foreign body 14 years previously, during her childhood. The patient's ages extended from 8 months to 46 years but were mostly between 1 and 3 years; apparently an age period when children are inexpert at eating and especially at chewing peanuts. Recovery

*From the Department of Broncho-Esophagology, Ste-Justine Hospital, Montreal, Canada. Presented on the International Night Program, 13th Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 5, 1947.

was complete except in three cases. These three patients were in such poor condition when they were brought to us that recovery could not be expected. These three patients were the only ones who required tracheotomy to permit the greatest freedom of manipulation and the greatest possibility of survival.

We have been able to observe every possible degree of bronchial obstruction, complete or incomplete brought about by the presence of these foreign bodies, as well as the secondary suppuration, emphysema and obstructive atelectasis. In about ten of these patients there was no history of a foreign body; in several of these and in a few others radiological examination was negative or showed very little to suggest a foreign body as a possible causal agent. The others mentioned vaguely that choking had occurred after placing a foreign body in the mouth. Only half of the patients sought or were brought for treatment during the first twelve days following the accident. At least 25 were admitted to hospital after a period varying from 12 days to 14 years after the entrance of the foreign body into the airways; in most cases the delay was caused by delay in diagnosis.

The nature of the foreign bodies has been varied enough:

I. Thirty vegetable or organic bodies (60 per cent of the total) of which 22 were peanuts (44 per cent of the total): the remains of a large dried bean, a timothy top, a fragment of a nut shell, two fragments of bone, two teeth, a chip of wood, a segment of pericarp of apple.

II. Sixteen metallic bodies or 32 per cent of the total: 4 open safety pins (plain and fancy); 2 common pins; 1 metal eraser holder from a pencil; the tubular portion of a tracheotomy cannula; the shell of a 22 rifle bullet; a part of a screw; the flexible part of a suction apparatus; a triangular piece of sheet iron; a ¾ inch nail; 2 carpet tacks; the shell of a fuse; the tab of a zipper.

III. Other foreign bodies: An oval wooden bead from a rosary; a large glass bead from a necklace; a piece of Celluloid from a toy.

According to Jackson 99 per cent of peanuts are removed from children. In that series I also have found only one peanut in an adult, a woman of 46 years. If one excepts this patient the ages extend from 15 months to 10 years, and are mostly between 1 and 3 years. These two years furnish our largest contingent for this type of foreign body, that is 77 per cent of all peanut cases, namely 17 out of 22.

INHALED PEANUTS:

Length of Sojourn: 4 months in the adult; 2 days to 2 months in the children.

ORGANIC FOREIGN BODIES:

No.	*Name*	*Age*	*Foreign Body*	*Location*	*Length of Sojourn*	*Main Symptoms*
51	Noel M.	19 months	4 fragments of a dried bean	Trachea, right and left bronchi	3 weeks	Fever, stridor, indrawing, cyanosis.
52	Francoise D.	22 months	Segment of pericarp of apple	Larynx	2 months	Laryngeal stridor, indrawing, dyspnea, aphonia.
53	Denis G.	20 months	Timothy top with spike	Left lower lobe bronchus	3 months	Juicy cough, fetid breath.
54	Marcel L.	8 years	Fragment of a hazel nut shell	Right lower lobe bronchus	6 months	Spiking fever, cough, septicemia, bad general condition.
55	Mrs. Aimé P.	35 years	Fragment of beef bone	Left lower lobe bronchus	15 days	Painful cough, distress, fever.
56	Claude L.	2 years	Chip of wood	Left lower lobe bronchus		Cough.
57	Unice S.	7 years	Molar tooth	Right main bronchus	3 weeks	Cough, fever, dyspnea.
58	Mrs. John C.	43 years	Chicken bone (small vertebra of neck)	Right main bronchus	10 days	Spells of painful coughing, acute pain in right chest, fetid breath.
59	Marthe P.	7 years	Incisive tooth	Right main bronchus	18 days	Cough and fever. Signs of atelectasis at right base.

SIXTEEN METALLIC FOREIGN BODIES: 8 on right side 50 per cent
7 on left side 43 per cent

60	Réal M.	6 years	Metal eraser holder from a pencil	Left lower lobe bronchus	5 days	Slight cough only.
61	Dieudonne B.	36 years	Tubular portion of a tracheotomy cannula	Right main bronchus	10 days	Cough only.
62	Léonce L.	5 years	Shell of a 22 rifle bullet	Left inferior lobe bronchus	17 days	Spells of cough and choking.
63	Rolland L.	13 years	Open large collar pin	Larynx and trachea	24 hours	Cough and dyspnea.
64	Yvette V.	3 years	Part of a screw	Right lower lobe bronchus	21 days	Indrawing, spiking fever, exhaustion, critical condition.

No.	*Name*	*Age*	*Foreign Body*	*Location*	*Length of Sojourn*	*Main Symptoms*
65	Lucien F.	33 years	Flexible part of a suction apparatus	Bronchus of posterior segment of right upper lobe	2 days	Abcess case of right upper lobe.
66	Annette T.	21 years	Common pin	Right lower lobe bronchus	4 days	Cough, blood spitting.
67	Mrs. Gerard L.	34 years	Small open safety pin with calcium and phosphate deposits	Right lower lobe bronchus	10 years	Productive cough, hemoptysis. Frequent attacks of grippe.
68	Marcel B.	11 years	1 common pin with piece of cork	Trachea	2 days	Slight cough only.
69	Mrs. Paul B.	21 years	Open small collar pin	Left lower lobe bronchus	14 years	Productive cough, many ounces of pus per 24 hours, septic condition. Complete carnification and massive bronchiectasis of left lower lobe.
70	Roland M.	2 years	Triangular piece of sheet iron	Larynx	12 months	Very marked dyspnea, indrawing and cyanosis. Critical condition.
71	Jacqueline C.	3 years	¾ inch nail	Left main bronchus		Intermittent cough.
72	Jean-Guy D.	21 months	2 carpet tacks	Right main bronchus	48 hours	Dyspnea, wheezing, cough and distress. Fever.
73	Jacques B.	8 months	Open fancy stick pin	Larynx and trachea	24 hours	Dyspnea, indrawing, aphonia, cyanosis.
74	Jacqueline R.	5 years	Shell of a fuse	Right main bronchus	1 week	Slight cough only.
75	Jacqueline B.	6 years	Tab of a zipper	Right main bronchus	1 week	Cough.
OTHER MISCELLANEOUS FOREIGN BODIES:						
76	Aline D.	10 years	Piece of celluloid from a toy	Larynx	24 hours	Marked stridor, indrawing and dyspnea. Aphonia.
77	Claude M.	2 years	Oval wooden bead from a rosary	Right main bronchus	24 hours	Signs of obstructive emphysema of left lung. Slight wheezing.
78	Yves 1.	4 years	Large glass bead from a necklace	Left main bronchus	18 hours	Signs of obstructive emphysema of left lung. slight wheezing.

Location: 10 in the right main bronchus (45 per cent); 4 in the left main bronchus (19 per cent); 2 in the right inferior lobe bronchus (9 per cent); 2 in the left inferior lobe bronchus (9 per cent); 3 in the trachea (13 per cent); 1 in the left superior lobe bronchus.

Clinical Signs in Children: Asthmatoid breathing, rhonchi and obstructive emphysema in most of the patients; indrawing of the chest, dyspnoea, cyanosis in 3 patients.

Radiological Findings: In about 50 per cent of the cases of peanuts, during expiration, emphysema of the obstructed lung; but there was displacement of the mediastinum towards the non-obstructed side, in a few cases only; atelectasis of the obstructed lobe or lung with displacement of the mediastinum towards this side was noticed in only a small number of children.

In a certain number of cases: entirely negative findings.

In the adult, whom I use for purposes of comparison, and who kept her foreign body for 10 months, there was chronic suppuration which could have passed for a bronchiectasis or tuberculosis. For this reason this patient had been under observation in a provincial sanatorium for several weeks and from there was sent to us for bronchoscopic examination.

I shall now outline briefly a few particularly interesting case histories: 1) A timothy top was found in the left inferior lobe bronchus in a child of 20 months. Putrid suppuration of the left inferior lobe of 3 months duration: cause unknown. The top is complete with a fairly long piece of the spike. This case was reported at length in Les Annales Médico-Chirurgicales de l'hospital Sainte-Justine (1941).

2) A section of the pericarp of an apple was suspended between the vocal cords of a child for 2 months. This child of 22 months showed stridor with inspiration, a croupy cough and dyspnoea simulating the syndrome of infectious or contagious laryngitis.

3) Half the shell of a hazelnut was removed from the right inferior lobe bronchus. It had been inhaled 6 months previously. This was a prize discovery for bronchoscopy because the clinical picture was that of a pleurisy with effusion or a pneumonia. It actually was simply a lobar atelectasis from obstruction.

4) Removal of 9 metallic foreign bodies, 4 in children:

(a) Part of the metallic band of a pencil. A boy of 6 years: length of sojourn 5 days in the left bronchus. A little cough only and a few rhonchi.

(b) A long safety pin, open, point up, between the vocal cords. Length of sojourn 24 hours. A boy of 13 years. Symptoms: cough and dysphonia.

(c) The body of a screw lodged in the left bronchus for 21 days. Child of 3 years. On admission general condition critical, indrawing of the chest, high fever, exhaustion.

(d) The shell of a 22 caliber projectile lodged for 12 days in the bronchial tree of a boy of 5 years. (Case reported in Les Annales Médico-Chirurgicales de l'hospital Sainte-Justine, 1940).

Five foreign bodies in the bronchi in adults:

1) Open safety pin, point up, in the left lower lobe bronchus on the left side, in a woman of 21. This pin had been inhaled 14 years previously when the patient was 7 years of age. A left pleural empyema had been drained at the age of 11 years, a fistula followed and persisted for 6 months. Copious expectoration daily since the fistula closed. Signs of putrid suppuration and toxaemia. To sum up she looked so much like a toxic case of tuberculosis that the Chest Specialists had forbidden her to nurse her baby and even isolated her from her child.

2) A tracheotomy cannula in a man suffering from cancer of the larynx—age 36 years, lodged in the right bronchus for 10 days. Transitory purulent bronchitis with localized ulcerations and without signs of obstruction. (Reported in the Transactions of The American Association of Broncho-Oesophagology, 1941).

3) The end of a metallic suction apparatus in the right superior lobe. This patient was 33 years of age and had a pulmonary abscess in this lobe.

4) An open safety pin in a woman of 41 years. This object had been lodged in the right inferior lobe bronchus for 12 years and was discovered when a radiograph was made on account of hemoptysis. (Presented before La Société de Phtisiologie de Montréal in 1941 and published in the American Annals of Oto-rhino-laryngology).

5) A fragment of beef bone in a workman, 35 years of age, lodged in the left bronchus for 15 days. Painful cough, fever, foetid expectoration. No abnormal x-ray shadows other than such as to suggest a peri-bronchial reaction at this level.

SUMMARY

On the whole we noticed that the symptoms and physical signs produced by foreign bodies could simulate those of bronchopulmonary diseases such as bronchial asthma, bronchopneumonia, tumors, infectious bronchitis and even tuberculosis.

It is thus most important that a complete study be made in every case in which the diagnosis of the bronchopulmonary dis-

ease has not been firmly established or confirmed. By a complete study we mean a complete past family and personal history, a careful physical examination, radiological studies, laboratory analyses and bronchoscopic exploration, followed if necessary, by complementary bronchographic studies.

Bronchoscopy, that is the bronchoscopic operation, is the only one that permits the removal of the cause of the obstruction, that is, the foreign body; or that provides the means of enlarging the lumen of a bronchus obstructed by a swollen mucosa, false membranes, debris, shreds of tissue or viscid secretions. It is in this way that the formerly appalling mortality in this type of illness has been reduced to the respectable figure of 2 per cent; 2 to 4 per cent only of foreign bodies in the bronchi are expelled by coughing; about 99 per cent can be removed by peroral bronchoscopy with a resultant complete recovery in 98 per cent of cases even if the prolonged sojourn of the foreign body has brought about extensive pathological changes.

In conclusion, we wish to point out that foreign bodies cause many deaths by asphyxia before the victim can get to a physician.

RESUMEN

En general, se observó que los síntomas y signos físicos producidos por cuerpos extraños pueden imitar los de enfermedades broncopulmonares, tales como asma bronquial, bronconeumonía, tumores, bronquitis infecciosa y aún tuberculosis.

Por consiguiente, es sumamente importante que se lleve a cabo un estudio completo de cada caso en el que no se ha establecido o confirmado firmemente el diagnóstico de la enfermedad broncopulmonar. Por un estudio completo queremos decir una historia complete del pasado de la familia y de la persona, un examen físico cuidadoso, estudios radiográficos, análises de laboratorio y exploración broncoscópica la que debe ser seguida, si es necessario, de estudios broncográficos complementarios.

La broncoscopía, es decir, la operación broncoscópica, es la única que permite la extracción de la causa de la obstrucción, a saber, el cuerpo extraño; o que ofrece el modo de ensanchar la luz de un bronquio obstruído por una mucosa hinchada, membranas falsas, desechos, fragmentos de tejidos o secreciones viscosas. De esta manera la mortalidad por esta enfermedad, anteriormente aterradora, ha sido reducida a la cifra respetable del 2.2 al 4 por ciento. Solamente un pequeño porcentage de los cuerpos extraños en los bronquios son expulsados mediante la tos; el 99 por ciento, más o menos, pueden ser extraídos mediante la broncoscopía oral, con la resultante reposición completa del 98 por ciento de los

casos, aún si la permanencia prolongada del cuerpo extraño ha producido alteraciones patológicas extensas.

En conclusión, deseamos indicar que los cuerpos extraños causan muchas muertes por asfixia antes de que la víctima pueda llegar donde el doctor.

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Fifteenth Annual Meeting
American College of Chest Physicians

The American College of Chest Physicians concluded a most successful meeting in Atlantic City, New Jersey, on June 5th. Over 900 doctors and their wives registered for this important meeting. A complete list of the registration is published in this issue. Reports of the activities of the meeting will be published in future issues of "Diseases of the Chest."

The scientific program was of the highest caliber which is a tribute to the Committe on Scientific Program whose members labored diligently in order to make this possible. The X-Ray Conference, conducted by Dr. M. C. Sosman of Boston, was one of the highlights of the meeting. It is unfortunate that more people could not be accommodated at the round table luncheons, most of which were sold out in advance of the opening dates of the meeting. The critical reviews on various aspects of chest diseases were well received and the evening devoted to the presentation of motion pictures drew a large audience.

More than 500 doctors and their wives attended the Annual Presidents' Banquet at which time guests from all of the other countries were introduced. Dr. Irving Willner, Chairman of the Committee on General Arrangements and all of the members of the New Jersey Chapter of the College are to be congratulated upon the excellent arrangements made for the meeting. Mrs. Irving Willner and the other members of the Women's Reception Committee are also to be congratulated for the hospitality extended to the doctors' wives who attended the meeting.

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U. S. Veterans Administration:
Roy A. Wolford, M.D., Washington, D. C.
U. S. Indian Service:
Arthur W. Dahlstrom, M.D., Rapid City, South Dakota

Governors of the College in U. S. Possessions

Alaska: A. Holmes Johnson, M.D., Kodiak
Hawaii: William F. Leslie, M.D., Hilo
Puerto Rico: Jaime F. Pou., M.D., Hato Rey

Governors of the College in Other Countries

Canada, Western Provinces:
Leslie Mullen, M.D., Calgary, Alberta
Philippine Islands: Manuel Quisumbing, M.D., San Pablo
Uruguay: Armando Sarno, M.D., Montevideo
Northern Italy: Attilio Omodei Zorini, M.D., Rome

All present Governors in other countries were re-elected.

ANNUAL MEETING REGISTRATION

State:	No. Registered
Alabama	6
Arizona	3
Arkansas	4
California	28
Colorado	15
Connecticut	9
Delaware	5
District of Columbia	14
Florida	11
Georgia	10
Illinois	55
Indiana	8
Kansas	4
Kentucky	18
Louisiana	5

	No. Registered	
Maine	4	
Maryland	26	
Massachusetts	22	
Michigan	17	
Minnesota	10	
Mississippi	2	
Missouri	14	
Nebraska	3	
New Mexico	1	
New Jersey	92	
New York	196	
North Carolina	7	
North Dakota	1	
Ohio	42	
Oklahoma	6	
Oregon	3	
Pennsylvania	94	
Rhode Island	5	
South Carolina	5	
South Dakota	1	
Tennessee	8	
Texas	18	
Utah	1	
Virginia	19	
Washington	1	
West Virginia	14	
Wisconsin	2	
TOTAL		809
United States Possessions:		
Alaska	1	
Hawaii	3	
Puerto Rico	7	
TOTAL		11
Other Countries:		
Argentina	7	
Belgium	1	
Brazil	1	
Canada	44	
Chile	4	
Cuba	7	
England	1	
India	1	
Mexico	8	
Republic of Panama	1	
Peru	2	
Philippine Islands	2	
Portugal	3	
South Africa	2	
Spain	1	
Switzerland	3	
Venezuela	1	
TOTAL		89
GRAND TOTAL		909

REPORT OF THE TREASURER

Financial Statement, May 31, 1949

The financial condition of the American College of Chest Physicians is more satisfactory than last year. We are not in the red in spite of the fact that expenses have doubled and the journal of the College is now being published twelve times a year instead of six times.

The actual cash income to 12/31/48 totalled			$60,891.23
This amount came from the following items:			
(1) New membership fees		$14,405.00	
(2) Dues		31,632.00	
(3) Sales:			
Advertising in Diseases of the Chest	$7,812.50		
Subscriptions	6,314.89		
Directory	30.00		
Making a total of		14,157.39	
(4) Interest on U. S. Govt. Bonds, Series "G"		557.50	
(5) Other income		139.34	
Making a total of			$60.891.23

The expenditures were as follows:

(1)	Salaries	$20,240.16	
(2)	Printing Diseases of the Chest	13,852.00	
(3)	Meeting Expenses	4,635.13	
(4)	Officers and Committee Expense	2,707.93	
(5)	Office Rent	4,349.19	
(6)	Printing and Engraving	1,999.88	
(7)	Postage and Shipping	2,042.86	
(8)	Travelling Expense	1,854.65	
(9)	Sales Discount	1,156.36	
(10)	Mailing and Posting of journal	1,914.47	
(11)	Office Expense	1,298.26	
(12)	Editors Expense, Diseases of the Chest	650.00	
(13)	Telephone and Telegraph	1,137.02	
(14)	Secretary to the president	300.00	
(15)	Secretary to Chairman, Board of Regents	300.00	
(16)	Membership Certificates	306.54	
(17)	Advertising Agency Expense	787.15	
(18)	Auditing	200.00	
(19)	Annual Award Expense	189.27	
(20)	Depreciation	361.74	
	Total Expense		$60,282.61
	Net Income		608.62

We have on reserve in U. S. Savings Bonds, Series "G" $33,800.00
of this amount $3,300.00 has accumulated from life memberships
$10,500.00 has accumulated for the Research Council
$20,000.00 has accumulated in the General Fund.

I am proud to report to you that the American College of Chest Physicians has gone far to promote the specialty of diseases of the Chest. The College has a very ambitious program and through its councils and committees great progress has resulted.

The American College of Chest Physicians is blessed with dreamers who

have practical dreams. The specialty of diseases of the chest is an established fact and it has come about only through the farsightedness and the vision of its officers and members.

However, we cannot sit on our laurels and feel happy about our accomplishments. A lot of work has yet to be done.

Any project that is undertaken by the College requires additional funds. Your officers do not desire to tax the members with prohibitive dues. There are other ways and means of raising additional funds to take care of our ambitious projects.

To do this we must:

(1) Obtain advertisements for Diseases of the Chest and support its advertisers.
(2) Let as many of us as possible, and preferably all of us, obtain life memberships so that more income may accumulate from this fund.
(3) Let us support the Research Council by the raising of funds among our wealthy philanthropic friends.
(4) There are many wealthy patients that leave fortunes when they pass on. It may be possible to get any number of them to will a part of their estates to the American College of Chest Physicians or to any one of its special projects.
(5) The College has established a book service department. When any of us wish to purchase any book, we can purchase it through our organization so that the publishers' discount may be used by us to a greater advantage.

In conclusion I wish to add that the LaSalle Auditing Company of Chicago checked our books and found them in good order.

Minas Joannides, M.D.

REPORT OF THE HISTORIAN

June 1, 1948 - June 1, 1949

Mr. President, Fellows of The American College of Chest Physicians and guests:

The constant labour and marvelous effort that have been given by physicians from the most distant past to our own time have revealed the truths of medicine as an art and as a science.

We pause at this time to pay respect and honour to our fellow physicians who devoted their lives to the application of these truths in the relief of pain and suffering of the afflicted and have now passed to their reward. It is fitting and proper that we do this.

The names of those Fellows of the American College of Chest Physicians who have passed to their reward since our last meeting are:

Dr. Lloyd H. Patterson, San Fernando, California.

Dr. Vera Viola Norton, Waverly, Iowa.

Dr. I. R. Fox, Eugene, Oregon.

Dr. Frank R. Wheelock, Scranton, Pennsylvania.

Dr. Eustace T. Goff, Vienna, West Virginia.

Dr. Burton W. Rhuberry, Ypsilanti, Michigan.

Dr. Harry S. Newman, Yonkers, New York.

Dr. Berthold S. Pollak, Jersey City, New Jersey. A pioneer worker in the field of tuberculosis in the State of New Jersey. A physician in honour of whom the Berthold S. Pollak Hospital was named.

Dr. Victor Cullen, Baltimore, Maryland. A pioneer and leader in tuberculosis work in the State of Maryland. President-elect of the National Tuberculosis Association, he stepped aside from the honour of assuming the office of President because of ill health.

Dr. Clemente Ferreira, Sao Paulo, Brazil. Dean and pioneer worker in the field of tuberculosis in his country.

Dr. Robert O. Brown, Santa Fe, New Mexico. Governor of the American College of Chest Physicians for the State of New Mexico.

Sir Sidney Valentine Sewell, Melbourne, Australia. A student and teacher, a physician and a world traveler, possessed of the kindliest disposition imaginable which endeared him to all his patients and friends. His early studies were in large measure devoted to neurological subjects but later he became interested in the study and treatment of tuberculosis, waging a never ending fight against this scourge. He played an important role in presenting to his government the tuberculosis problem and in setting the policy and plans for its eradication in his country. The Sir Sidney Sewell Pathological Wing of the New Tuberculosis Sanatorium at Watsonia is a memorial to his constancy and to his unselfish work of love.

We pay our respect and do honour to the memory of these departed physicians. But, in a larger sense, we pause to rededicate and reconsecrate our efforts, our talents and our lives to the continuation of the never ceasing and unselfish work of charity, love and mercy, begun in the distant past and continued by physicians from time immemorable.

The world may little heed what we say, but time nor faulty memory can erase the glorious advances made through their constancy and never-ending search for the truth. It is for us to take from these honored physicians of the past an increased devotion to the cause of humanity for which they gave a full measure of devotion, that we shall resolve that their constancy, that their unselfishness and that their labour of love shall not have been in vain, that the welfare of our patients shall always be first in our minds, that the truths of medicine shall blossom forth in a new light and shall remain untrammeled.

William A. Hudson, M.D.

POSTGRADUATE COURSE IN BRONCHOESOPHAGOLOGY

The University of Illinois, College of Medicine, announces a postgraduate course in Bronchoesophagology which will be supervised by Doctors Paul H. Holinger and Albert H. Andrews.

This course will be held from September 19 to October 1, 1949. For application forms and further information communicate with the Postgraduate Division (Otolaryngology), University of Illinois, 1853 West Polk Street, Chicago 12, Illinois.

College Chapter News

ARIZONA CHAPTER

The annual meeting of the Arizona Chapter of the College convened at the Pioneer Hotel in Tucson on May 10, in conjunction with the state medical society meeting. The following officers were elected:

Kent H. Thayer, M.D., Phoenix, President
John W. Stacey, M.D., Tucson, Vice-President
Leslie B. Smith, M.D., Phoenix, Secretary-Treasurer

The President of the Chapter has appointed Dr. Dan Mahoney of Tucson as Chairman of the Medical Education Committee, Dr. John L. Cogland, Chairman, and Drs. N. K. Thomas and Hilton J. McKeown, members of the Program Committee, and Drs. Edward J. Nagoda and Bertram L. Snyder to the Membership Committee.

ARGENTINE CHAPTER

The Argentine Chapter of the College has instituted a postgraduate scholarship at the Cátedra de Patologia y Clínica de la Tuberculosis, Buenos Aires for doctors from the interior of the country who are interested in postgraduate training in diseases of the chest. Dr. Raul F. Vaccarezza, Governor of the College, is professor at the institute. Dr. Justo Lopez Bonilla, President of the Argentine Chapter, and Drs. Gumersindo Sayago, Raul Vaccarezza and Agustin Caeiro constitute the board of examiners. The scholarship pays 400 pesos per month (Arg.) for a period of six months.

ILLINOIS CHAPTER

At the annual meeting of the Illinois Chapter of the College held at the Palmer House, Chicago, on May 15, the following officers were elected:

Darrell H. Trumpe, M.D., Springfield, President
Edwin R. Levine, M.D., Chicago, Vice-President
Charles K. Petter, M.D., Waukegan, Secretary-Treasurer

A resolution was adopted that the chapter go on record as recommending to the state medical society that a Section on Diseases of the Chest be established in the scientific assembly. It was pointed out that the New York State Medical Society and the New Jersey State Medical Society now have such sections.

Plans were discussed for the annual postgraduate course in diseases of the chest to be presented at the Hotel St. Clair, Chicago, September 19-23. It was recommended that greater emphasis be given to cardiac conditions in the next postgraduate course.

The meeting was attended by Dr. James H. Stygall, Regent of the College for the district, and Dr. Italo Volini, the Governor of the College for the state of Illinois. Dr. Andrew L. Banyai, Regent of the College for District No. 10 was one of the guest speakers.

MICHIGAN CHAPTER

On May 4 the members of the Michigan Chapter of the College played host to the members from outside the state at a meeting in Detroit. At 6:00 p. m. a cocktail party was held which was followed by dinner at the University Club. Fifteen members and four guests were present.

MINNESOTA CHAPTER

The Minnesota Chapter of the College held its annual meeting in St. Paul on May 9. Dr. John F. Briggs, St. Paul, was elected President of the chapter for the year 1949-50 and Dr. Karl H. Pfuetze, Cannon Falls, was elected Secretary-Treasurer.

At the request of Dr. Hilbert Mark of the State Department of Health, the Chapter decided to establish a speakers bureau on chest diseases. These speakers will be called upon by Dr. Mark to address county medical society meetings, luncheon clubs and various lay groups regarding the different phases of tuberculosis control work in the state.

The Chapter voted to collaborate with the University of Minnesota in sponsoring a postgraduate course on diseases of the chest which will be held at the Center for Continuation Study on the University of Minnesota Campus in the fall of the year.

WISCONSIN CHAPTER

Dr. Leon H. Hirsh addressed the March meeting of the Metropolitan Milwaukee Section of the Wisconsin Chapter on the subject of "Acroerythrosis." At the April meeting of the Metropolitan Milwaukee Section, Dr. Karl E. Kassowitz presented a talk on "The Use of Streptomycin in Children."

College News Notes

Dr. Edwin R. Levine, formerly Director of Chest Service at Michael Reese Hospital, Chicago, and Medical Director of Winfield Sanatarium, Winfield, Illinois, has opened offices for the private practice of medicine at 109 North Wabash Avenue, Chicago. He will specialize in the treatment of diseases of the chest.

Dr. Jose Ignacio Baldo, Caracas, Venezuela, Regent of the College, has been appointed a member of the Committee on Tuberculosis of the World Health Organization. The next meeting of the committee will be held in Copenhagen, Denmark on July 26 of this year.

Dr. Chester G. Crist, Gettysburg, Pennsylvania, Gettysburg College physician for nearly 35 years, was awarded the "meritorious service award" for the past year by Gettysburg College. Dr. Crist is also jail and county home physician, a post which he has held for the past 15 years, and has served as County Medical Director and Chief of the State Tuberculosis Clinic for the past 25 years.

Dr. Robert H. Marks has returned to his position as head of the Bureau of Tuberculosis Control of the Territorial Department of Health in Hawaii after a year's absence.

The Tuberculosis Committee of the Iowa State Medical Society for 1949 consists of the following members: Raymond J. Harrington, M.D., Sioux City, Chairman; John C. Parsons, M.D., F.C.C.P., Des Moines; J. Carl Painter, M.D. F.C.C.P., Dubuque; Leon J. Galinsky, M.D., F.C.C.P., Des Moines; Ralph E. Smiley, M.D., Mason City; William Spear, M.D., F.C.C.P., Oakdale; and Daniel R. Webb, M.D., Cedar Rapids.

Announcement

FALL POSTGRADUATE COURSES IN DISEASES OF THE CHEST

Chicago, Illinois — September 19-23

St. Clair Hotel — 5 days — Tuition $50.00

Sponsored by the Illinois Chapter, American College of Chest Physicians.

Minneapolis, Minnesota — October 20-22

Center of Continuation Study, University of Minnesota

3 days — Tuition $20.00

Presented by the University of Minnesota under the sponsorship of the Minnesota Chapter, American College of Chest Physicians.

New York, N. Y. — November 14-19

Hotel New Yorker — 5 days — Tuition $50.00

Sponsored by the New York State Chapter, American College of Chest Physicians.

San Francisco, California — December 5-9

Postgraduate Extension Building, University of California

5 days — Tuition $50.00

Presented by the California Chapter, American College of Chest Physicians, in cooperation with the University of California Medical School and Stanford University School of Medicine.

For further information please write to the Executive Offices,
American College of Chest Physicians,
500 North Dearborn Street, Chicago 10, Illinois